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PATHOLOGY IN SURGERY

By Edwin F. Hirsch, Ph.D., M.D., *Director of Henry Baird Favill Laboratory and Pathologist, St. Luke's Hospital, Chicago; Research Associate and Associate Professor Emeritus, Department of Pathology, University of Chicago*

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CONTENTS, DECEMBER

Surgical Therapy for Cholecystitis: Observations Based on a Review of Four Hundred and Seventeen Cases. C. D. KNIGHT, M.D., B. JOHNS, M.D., AND J. A. HENDRICK, M.D., *Shreveport, La.*....1137

Simultaneous Small and Large Bowel Obstruction Resulting from Chronic Regional Enteritis. J. L. HERRINGTON, JR., M.D., W. R. Cate, JR., M.D., AND A. B. LIPSCOMB, M.D., *Nashville, Tenn.*....1149

Acute Gastric Dilatation. W. J. GILLESBY, M.D., AND J. R. WHEELER, M.D., *Hines, Ill.*....1154

Treatment of Peripheral Arteriosclerotic Disease by Arterial Homografting. D. J. AUSTIN, M.D., AND J. E. THOMPSON, M.D., *Dallas, Texas*....1163

Necrotizing Panarteritis: A Complication of Repair of Coarctation of the Aorta. J. B. GROW, M.D., C. V. DEMONG, M.D., AND W. R. RUNDLES, M.D., *Denver, Colo.*....1168

Clinical Experiences with Prefabricated Nylon Artery Grafts. W. S. EDWARDS, M.D., AND J. S. TAPP, PH.D., *Birmingham, Ala.*....1174

Rupture of Abdominal Aortic Aneurysm into Inferior Vena Cava: Successful Surgical Repair. H. B. SHUMACKER, JR., M.D., AND J. JONTZ, M.D., *Indianapolis, Ind.*....1182

Acute Appendicitis in the Elderly Male. W. J. GILLESBY, M.D., *Hines, Ill.*, AND C. M. O'REILLY, M.D., *Palos Park, Ill.*....1186

Late Sequellae of Traumatic Eventration of the Diaphragm and Their Surgical Management. E. R. MAURER, M.D., *Cincinnati, Ohio*....1194

Surgery in Pulmonary Tuberculosis: The Problem of the Poor-Risk Patient. N. H. KRAEFT, M.D., AND L. O. LINTON, M.D., *Tallahassee, Fla.*....1207

Rupture of the Small Intestine Through the Intact Abdominal Wall Without Associated Intraperitoneal Injury. J. T. DUNCAN, JR., M.D., *Memphis, Tenn.*....1215

Successful Embryonic Parathyroid Tissue Transplant for the Treatment of Intractable Postoperative Parathyroid Tetany. H. L. RIGDON, M.D., AND W. R. MEAD, M.D., *Florence, S. C.*....1222

Intravenous Cholecystography and Cholangiography Using Cholografin. C. E. BICKHAM, JR., M.D., *Washington, D. C.*....1225

Editorial: Not for Ourselves Alone. C. W. MAYO, M.D., *Rochester, Minn.*....1233

Book Reviews....1234

Index to Volume XXII....1237

The American Surgeon

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SURGICAL THERAPY FOR CHOLECYSTITIS: OBSERVATIONS BASED ON A REVIEW OF FOUR HUNDRED AND SEVENTEEN CASES

CHARLES D. KNIGHT, M.D., BARRON JOHNS, M.D., JOHN A. HENDRICK, M.D.

Shreveport, La.

Surgical treatment of the inflammatory diseases of the gallbladder has become accepted as the most efficacious therapy that presently can be offered. This acceptance has come inevitably from the good results that one encounters in his own experience as well as those reported throughout the literature.

Periodically, however, it seems that all clinicians should analyze their own results with a given form of therapy to ascertain if, in their hands, it measures up to the accepted standards.

With this in mind a study was undertaken with two primary objectives: (1) to review our experience with cholecystitis for the purpose of analyzing the results and comparing them with some suitable yardstick, and (2) to compare the different age groups of patients with cholecystitis to see if similar treatment in all groups was justified.

Material: During the 15 year period from January 1940 through December 1954, 417 patients were operated upon at the Highland Clinic for gallbladder disease. These clinical records were carefully reviewed and the data obtained are the basis of this report. For purpose of comparison, the data on patients 60 years of age and older were tabulated separately and will be referred to as Series A. The remaining data concerning patients who were 59 years of age and younger will be referred to as Series Y.

CLINICAL FINDINGS

The total series was comprised of 337 female and 80 male patients. There were 3 Negro patients and the remainder were white. The greatest age incidence occurred during the sixth decade (table I). The youngest patient was 16 years of age and the oldest was 85.

In the group of patients who were 60 years of age and older (Series A) the average over-all duration of symptoms prior to hospital admission was 47 months.

From the Department of Surgery, Highland Clinic, Shreveport, La.

TABLE I

Age distribution

Age, yrs.	Patients, no.
10–19	2
20–29	24
30–39	67
40–49	91
50–59	111
60–69	90
70–79	26
80–90	6

TABLE II

Historical data: comparison of symptoms in two series

Symptoms	Series A (122 patients) Age 60 yrs. and over, per cent	Series Y (295 patients) Age 59 yrs. and under, per cent
Pain	84	93
Fever	26	9
Chills	9	2
Jaundice	14	8
Colic	68	67
Previous attacks	79	85

For the group of patients under 60 years of age (Series Y) the average duration of symptoms was 34.4 months.

Table II shows a comparison of the historic data recorded in the two groups of patients, and the differences noted do not seem remarkable. As might be anticipated from one's clinical experience, pain was the commonest symptom noted. A history of chills and/or fever was not common, particularly in the younger series. Jaundice was noted by the patient in 14 per cent of the older group and 8 per cent of the younger. The percentage of patients in the two groups having a history of colic was almost identical, and a high percentage of both groups had had previous attacks of pain.

TABLE III

Historical data: other diseases concurrently present

Diseases	Series A (122 patients) Age 60 yrs. and over	Series Y (295 patients) Age 59 yrs. and under
Coronary artery disease	4	1
Diabetes	5	2
Hypertensive cardiovascular disease	5	2
Cerebrovascular accident	1	0
Duodenal ulcer	2	9
Regional ileitis	1	0
Carcinoma sigmoid	0	1
Inguinal hernia	0	1

TABLE IV

Physical findings

Finding	Series A (122 patients) Age 60 yrs. and over, per cent	Series Y (295 patients) Age 59 yrs. and under, per cent
Tenderness	73	78
Rigidity	19	13
Palpable gallbladder	8	3
Mass	11	6
Elevated temperature	20	15
Obesity	13	19
Jaundice	8	5
Hypertension	18	2
Fibrillation	.8	.4

In table III there is a breakdown of data obtained concerning the presence of other unrelated diseases in the patients in the two age groups. Fourteen per cent of the patients in the older series had had some unrelated disease, while only 5 per cent of the younger group gave such a history.

An analysis of the physical findings in table IV shows little difference in the two groups. Tenderness was by far the most consistent finding and, as might be expected, the incidence of hypertension was greater in the older age group.

Laboratory Data: A study of the laboratory data in this series showed leukocyte counts ranging from normal to 27,750 per cubic millimeter of blood. Sixty per cent of the patients in Series A (60 years of age and older) had normal counts (less than 9,000) while 71 per cent of the patients in Series Y (under 60 years of age) had normal counts on admission to the hospital. The leukocyte counts in the cases of acute cholecystitis were similar in both series. The average leukocyte count of the acute cases in the older patients was 13,000 with 81 per cent polymorphonuclear leukocytes compared to 11,500 with 78 per cent polymorphonuclear leukocytes in the younger group. In the total series there were 60 patients with an elevated serum bilirubin: 27 in the aged group and 33 in the younger series.

Impaired liver function, as measured by the cephalin flocculation and bromsulphalein tests, was noted in 12 patients. Eight of these were 60 years of age or older and 4 were in the younger group.

Roentgenographic Studies: Table V shows an analysis of the roentgenographic

TABLE V

Roentgenographic studies

	Patients, no.
Nonfunctioning gallbladder	94
Nonfunctioning gallbladder with stones	15
Stones on flat plate of abdomen	9
Functioning gallbladder with stones	117
Poorly functioning gallbladder	17
Normally functioning gallbladder—no stones	15

findings on those patients in the total series who had roentgenograms available for review. These findings then were correlated with the operative findings to ascertain the degree of accuracy of the interpretation of the roentgenograms. Of 94 patients with nonfunctioning gallbladders, 5 failed to demonstrate stones or some other evidence of disease. Of 168 patients whose roentgenograms were interpreted as demonstrating gallstones, with or without gallbladder function, 3 were found to have no stones or pathologic findings in the biliary tract at the time of operation. In 17 patients a roentgenographic report of poorly functioning gallbladder was obtained and exploration revealed normal biliary tracts in 5. Of those 15 patients explored for symptoms suggestive of biliary tract disease who had normally functioning gallbladders on preoperative cholecystograms, 2 were found to have stones and gallbladder disease.

Comment: While the preponderance of females with cholecystitis has been frequently reported, the ratio to males with the disease varies considerably among the reported series. The sex incidence here favored females more than 4 to 1. A recently reported large series from the Lahey Clinic[1] showed a sex ratio favoring females 2 to 1.

The great preponderance of white patients in this series is not surprising since there is a great discrepancy between white and Negro admissions in our hospital. There is general acceptance that the incidence of gallbladder disease is less in the Negro than in the white race but little emphasis have been placed on this fact. In a recent study of 864 patients with gallbladder disease from both private and charity hospitals in this area, Mathews[5] found that the ratio of gallbladder disease was 5 times as prevalent in the white as in the Negro race. His study also showed the rarity of the disease in Negro males since he found only 11 such patients out of his series of 864. All 3 Negro patients in the series discussed in this paper were females.

The greatest age incidence in this series was the sixth decade, the same as reported in two Lahey Clinic series.[1] Mathews[5] noted a difference in the peak incidence in his series for patients with chronic cholecystitis (the fifth decade) and with acute cholecystitis (two decades later). This has been a frequent clinical observation and is believed to reflect the importance of long-standing "*silent*" stones as a causative factor in acute cholecystitis.

The large number of older patients whose ages ranged to 85 years is in keeping with reports of others that indicate that as the mean age of the population rises, we may expect an increase in cholecystitis. Strohl and Diffenbaugh[7] have pointed out that biliary tract disease represents the most frequent single cause for abdominal surgical intervention in the geriatric patient.

While the youngest patient in this series was 16 years, there is some evidence that cholecystitis is either becoming more frequent or is being recognized more often in the pediatric group of patients. Forshall and Rickham[2] recently reported 6 cases of gallbladder disease in females with ages ranging from 2½ to 12 years. They called attention to the fact that mild attacks of cholecystitis in children usually escaped attention and that many cases of cholecystitis first recognized in middle life had their inception in childhood.

Most studies of gallbladder disease have demonstrated the simultaneous presence of other pathologic conditions and the increased incidence in the aged group of patients might be anticipated. Diabetes, hypertension, arteriosclerotic heart disease and duodenal ulcer were the four most common associated conditions present here, and a similar high frequency of occurrence has been reported by others.[3] It has been suggested that the high incidence of preoperative diagnosis of duodenal ulcer in patients with cholecystitis might be erroneous due to the deformity of the duodenal cap that could be caused by contiguous inflammation of the gallbladder. In this series, at least, a high incidence of duodenal ulcer was found at laparotomy, and it has been our experience that the two lesions, cholecystitis and duodenal ulcer, are frequently associated. For this reason, in patients with upper gastrointestinal symptoms, both organs should be investigated preoperatively as well as at the time of operation.

Laboratory values in this series were not remarkable but it is of interest that the leukocyte counts in the patients with acute cholecystitis were of the magnitude that might be expected in cases of acute appendicitis. This was true for differential as well as total counts. As in patients with acute appendicitis a normal leukocyte count did not rule out acute inflammation of the gallbladder.

The continued accuracy of roentgenographic study of the gallbladder is again demonstrated in this study. A report of nonfunctioning gallbladder was synonymous with disease of the gallbladder in 95 per cent of the patients. A positive report of gallstones was erroneous in 1.7 per cent of the patients seen. A report by our radiologist of a poorly functioning gallbladder indicated pathologic findings in 70 per cent of the patients who were explored. In this series, as in others, gallstones were infrequent in patients with normal cholecystograms, but they did occur.

PATHOLOGIC FINDINGS AND SURGICAL PROCEDURES

A review of the surgical pathologic findings (table VI) shows an increased incidence of acute cholecystitis in the older age group of almost 2 to 1 and a greater occurrence of adenocarcinoma of the gallbladder. The incidence of presence of stones was 88 per cent in both groups of patients.

Table VII shows that cholecystectomy was performed in 86 per cent of the older patients and in 98 per cent of the younger ones. The alternative procedure

TABLE VI

Pathologic findings

Diagnosis	Series A (122 patients)		Series Y (295 patients)	
	Number	Per cent	Number	Per cent
Acute cholecystitis	25	20	34	11
Subacute cholecystitis	7	5	7	2
Chronic cholecystitis	90	73	254	86
Cholelithiasis	108	88	261	88
Adenocarcinoma of gallbladder	3	2	0	0

TABLE VII

Surgical procedures

Operation	Series A (122 patients)		Series Y (295 patients)	
	Number	Per cent	Number	Per cent
Cholecystectomy	106	86	290	98
Partial cholecystectomy	2	1.6	1	.3
Cholecystostomy	14	11	4	1.3
Choledochotomy	40	32	60	20
Choledocholithotomy	27	22	25	8
Previous cholecystostomy	6	5	9	3

most frequently utilized was cholecystostomy in 11 per cent of the older series and 1.3 per cent of the younger group. These data also reveal a higher incidence of exploration of the common duct in the geriatric series as well as an increase in the number of common duct stones found. Five per cent of the older patients and 3 per cent of the younger patients had a previous cholecystostomy prior to definitive cholecystectomy in this series.

Nine patients had major surgical procedures (exclusive of appendectomy) in addition to surgery of the biliary tract. Three of these had resections of the right colon for carcinoma, 3 had a subtotal gastrectomy for duodenal ulcer, 2 had a gastroenterostomy and 1 had an ileocolostomy for regional ileitis.

At operation there were a number of miscellaneous findings, some related to cholecystic disease and others entirely unrelated. There were 4 patients with fresh perforations of the gallbladder and evidence in 4 of old perforations without fistula formation. There were 7 patients with cholecysto-enteric fistulas, 4 involving the duodenum and 3 involving the colon. There was one instance of a perforated cystic duct. The most common incidental findings were duodenal ulcer and pancreatitis, each of which was found in 8 patients. Other findings included 3 patients with carcinoma of the colon, 3 with ampullary stricture, 2 with cirrhosis of the liver, 2 with polycystic kidneys, 2 with metastatic carcinoma and one each with regional ileitis, benign hepatic cyst, tuberculosis of the liver, primary hepatoma, diaphragmatic hernia, ovarian cyst, gastric polyp and appendiceal abscess.

Comment: Glenn and Hays[3] have stressed that with increased age there is an apparent increased incidence of acute cholecystitis and an increased incidence of associated biliary tract neoplasia. This was true in this series; acute cholecystitis was twice as frequent in patients 60 years of age and older and all patients with carcinoma of the biliary tract were 60 years of age and older.

While cholecystostomy is not considered to be a definite procedure in the long-term treatment of cholecystitis and cholelithiasis, its choice by surgeons in 11 per cent of the patients 60 years of age and older in this series shows that it was believed to have a definite place in the treatment of patients with gallbladder disease in this series.

The advantages of cholecystostomy in the very ill or poor-risk patient or in the

TABLE VIII

Choledochotomy and choledocholithotomy: comparison of incidence with other reported series

Series (Years)	Choledochotomy, per cent	Choledocholithotomy, per cent
Reported series (1940–1954)	23.9	12.4
Lahey Clinic[1] (1950–1953)	36.7	10.4
Mayo Clinic[8, 9, 10] (1951–1953)	22.7	11.4

face of marked technical difficulties at the time of operation are well recognized and accepted. One must not lose sight of these advantages in his zeal to do a definitive procedure.

Three and one-half per cent of the patients in this total series had had previous cholecystostomy. This approximates the 3.7 per cent incidence of previous cholecystostomy reported by Colcock and McManus[1] in their 1,356 patients who required definitive cholecystectomy. These figures support the contention that most patients who require cholecystostomy will require subsequent cholecystectomy in order to satisfactorily complete their treatment.

The usual indications for exploration of the common duct are utilized in this clinic and in this total series, 23.9 per cent of the procedures were accompanied by exploration of the common duct. Common duct stones were found and removed in 12.4 per cent of the total number of patients. In table VIII this incidence of choledochotomy and choledocholithotomy is compared with recent reports from the Lahey[1] and Mayo Clinics.[8, 9, 10] Although there is some variation in the frequency of exploration of the common duct, it is interesting to note that the rate of stone recovery is almost identical in these reports.

The increased incidence of choledocholithiasis in the aged patients has been stressed by Glenn and Hays[3], Quigley[6], Welch[11] and others. This was observed in this series with a proved incidence of 22 per cent in patients 60 years of age and older compared with 8 per cent in patients 59 years of age and younger.

One of the problems encountered in abdominal surgery is the gallbladder containing stones when it is found in the course of exploration during surgery for other abdominal disease. In 9 patients in this series, this dilemma resulted in the surgeon removing the gallbladder in addition to the contemplated surgery, and in these patients the convalescence was uneventful.

The decision to remove the gallbladder under such circumstances is not always a simple one. Many authors have called attention to the hazard of acute cholecystitis in patients following the surgical treatment of unrelated disease and Glenn and Wantz[4] recently reviewed 18 such cases. Mathews[5] also has stressed the danger of leaving the diseased gallbladder. He stated that not only does usual postoperative treatment, such as withholding of food for several days and administration of opiates, set the scene for the development of acute cholecystitis, but should it develop, it is likely to be masked by drugs or mistaken for some less serious condition. This fact has been verified in Mathews' experience by the occurrence almost yearly of a postmortem examination on a patient with gangrenous cholecystitis arising from stones knowingly or unknowingly left at the time

of an abdominal operation for some other disease. A word of caution in this matter has been expressed by Colcock and McManus[1]. Of 16 deaths which occurred following biliary tract operations in their 1,356 patients, 5 occurred in patients who had had an additional major abdominal procedure at the time of cholecystectomy. In view of their experience, they advise careful evaluation before combination of cholecystectomy with another major abdominal procedure.

The 7 cholecysto-enteric fistulas encountered represent an incidence of 1.6 per cent in this series. This is similar to a 1.4 per cent incidence of internal biliary fistula reported by Colcock and McManus. These figures show the frequency of involvement of the colon and duodenum by inflammatory disease of the gallbladder. While the occurrence of fistulas in our series was in patients with long-standing biliary tract disease it is of interest that it was more frequent in the younger patients.

COMPLICATIONS AND MORTALITY

The nonfatal complications encountered in this series are tabulated in table IX. The most common of these in the order of their frequency were urinary cystitis, unexplained fever, pneumonia, thrombophlebitis and pulmonary embolism. The occurrence of complications was slightly greater in the aged group than in the younger group. The combined nonfatal complication rate in the total series was 11.5 per cent.

Forty-one per cent of the total patients were given postoperative antibiotics and most of these were used in patients 60 years of age and older.

In table X the mortality following operative procedures for cholecystitis in this series is summarized. There were 3 deaths in the entire series. These all occurred in patients in the older age group. Two were in patients who had had exploration of the common duct in addition to cholecystectomy and the other in a patient who had had cholecystostomy alone. The 3 deaths comprise a mortality rate in 122 older patients of 2.4 per cent as contrasted with no mortality in 295 patients of the younger ages. The mortality rate of the total series was 0.7 per cent.

TABLE IX

Nonfatal complications of biliary tract surgery

Complications	Series A (122 patients)	Series Y (295 patients)
Pulmonary embolism	3	2
Thrombophlebitis	1	6
Fever, unexplained origin	6	6
Cystitis	2	10
Upper respiratory infection	3	5
Pericarditis	1	0
Paroxysmal auricular tachycardia	0	1
Postoperative arthritis	0	1
Postoperative shock	0	1
Total	16 (13%)	32 (10.8%)

TABLE X

Mortality following operative procedures for cholecystitis

Procedures	Series A (122 patients)			Series Y (295 patients)		
	Cases	Deaths	Per cent	Cases	Deaths	Per cent
Cholecystectomy alone	69	0	0	231	0	0
Cholecystectomy plus choledochotomy	39	2	5	60	0	0
Cholecystostomy alone	13	1	7.6	4	0	0
Cholecystostomy plus choledochotomy	1	0	0	0	0	0
Totals	122	3	2.4	295	0	0

Analysis of Deaths: There were 3 deaths in this series and the circumstances of these cases are summarized briefly.

1. In 1940, a 65 year old white man who had had symptoms of gallbladder disease for 10 months and who was jaundiced was admitted to the hospital. After 48 hours of preparation the patient was operated upon and chronic cholecystitis, cholelithiasis and a cholecystoduodenal fistula were found. Cholecystectomy and choledochotomy were carried out but no stones were found in the common duct. The duodenal fistula was closed. The postoperative course was febrile and death ensued 48 hours after operation. No autopsy examination was made.

2. In 1949, a 72 year old white woman was admitted to the hospital 48 hours after the onset of symptoms of gallbladder disease. After preparation, cholecystostomy was performed for acute cholecystitis. The patient died 48 hours after operation and an autopsy examination revealed bilateral polycystic kidney disease with a polycystic liver.

3. In 1954, a 75 year old white woman was admitted to the hospital with acute cholecystitis and hypertension. After 48 hours of preparation and observation, cholecystectomy was performed and choledochotomy revealed an ampullary stricture. A transduodenal exploration was carried out and then a choledochoduodenal anastomosis. The postoperative course was febrile and on the sixth postoperative day she became comatose and died. Autopsy revealed acute liver necrosis and a lower nephron nephrosis.

Comment: The number of nonfatal complications seen in our total series is too small to allow very striking conclusions to be drawn. However, the incidence of occurrence here was similar to that reported by others.[1, 3] Also, as in other series[3], the incidence of complications was slightly higher in the aged group than in the younger patients. The effect of antibiotics on the complication rate and hospital course was revealing. The series started when antibiotics were unavailable but with their advent they were used rather routinely by some surgeons for a period of time so that over-all 41 per cent of the patients were given postoperative antibiotics. A survey of the patients shows no appreciable difference in complication rate, morbidity or mortality whether or not antibiotics were used. Currently, it is the practice not to use these drugs without specific indication, and their use has diminished considerably.

TABLE XI

Mortality rate: comparison with other reported series

Series (Years)	Number cases	Mortality rate, per cent
Reported series (1940–1954)	417	.7
Lahey Clinic[1] (1950–1953)	1,356	1.1
Mayo Clinic[8, 9, 10] (1951–1953)	3,581	.7

All deaths in this series were in patients over 65 years of age and all were complicated cases requiring something to replace, or something in addition to, cholecystectomy. Here, as in other series[1, 3], the mortality of patients over 60 years of age was more than double that of younger patients. A comparison of the over-all mortality rate of this series with the rates of two large series[1, 8, 9, 10] is shown in table XI.

It is apparent on reviewing the reported series of gallbladder disease that, while the mortality factors are many and varied, the prime difficulty lies not in patients with uncomplicated biliary tract disease who require cholecystectomy alone, and perhaps not even in patients whose surgical treatment necessitates the addition of choledochotomy where stones are not found in the common duct. The risk of this procedure, at least in some hands[10], has approximated that of cholecystectomy alone. Along with the complications of gallbladder disease comes the increase in mortality. In one year at the Mayo Clinic[10], it was reported that jaundice doubled the risk of the operation and that when stones were found and removed from the common duct, the risk increased to four times that of cholecystectomy alone. In the series reported by Colcock and McManus, 50 per cent of the deaths occurred in patients jaundiced at the time of surgery.

With such recurrently reported experience, every effort should be directed toward operating upon patients with cholecystic disease earlier in the course of that disease thus decreasing the likelihood of these later complications. The duration of symptoms of patients treated in this series suggests the long duration of the disease process before treatment and in many instances, before diagnosis was made.

Perhaps one of the greatest deterring factors in advising cholecystectomy in a patient with proved gallstones is advanced age. Many patients in whom a diagnosis of gallstones was made 5 to 10 years before, who were advised against operation at that time because of their advanced age, have required emergency biliary tract operation in this series. Glenn and Hays' study of the age factor in mortality rate in biliary tract surgery deserves wide dissemination. They stressed the peculiarities of gallbladder disease in elderly patients that increase the mortality—the increase in acute cholecystitis, the increase in choledocholithiasis, the possible absence of characteristic signs and symptoms in acute disease, and the increased incidence of neoplasia. (All these were noted in the older patients in this series.) Even more important, they showed that elective biliary tract surgery is tolerated well by the geriatric patient and that there is little difference in the complication and mortality rates in this group when compared to a younger group of patients. In contrast, they demonstrated a definite increase in mortality

and morbidity if the aged patient required emergency surgery. This seems to give a clear mandate to treat disease of the gallbladder when and where it is found and to avoid procrastination.

A review of results in this series, when compared with available yardsticks, shows the mortality and complication rates to be fairly acceptable. This seems best attributed to an over-all policy of conservatism, reflected in the use of cholecystostomy in poor-risk cases of acute cholecystitis, especially in the older age group, and in careful observation and preparation of most cases of acute cholecystitis. While we have not been opposed to early surgery for acute cholecystitis, most of these patients were seen later than 48 hours after onset and responded well to measures in preparation for cholecystectomy. Thus surgery usually was performed 5 to 10 days after the onset of symptoms.

While the mortality and morbidity rates might be difficult to improve, the incidence of perforations and fistulas of the gallbladder seem to be an indication for consideration of earlier surgery. In addition, the repeated finding of an acute pathologic process out of all proportion to the mild clinical findings, warns of the hazard of unwarranted delay before surgery.

Currently, it seems wise to us to evaluate each gallbladder problem individually and to plan the treatment according to the specific situation encountered. Some patients will require further diagnostic study, others time to improve their general condition, while still others are best handled by prompt surgical intervention.

SUMMARY

The clinical records of 417 patients who were operated upon for gallbladder disease at the Highland Clinic during the 15 year period from 1940 through 1954 have been reviewed and the data are herein reported. The patients were divided into those older and those younger than 60 years of age for the purpose of comparison.

Cholecystectomy was performed in 86 per cent of the older patients and in 98 per cent of the younger ones. Cholecystostomy was the most common alternative procedure employed.

Exploration of the common duct was performed for the usual indications and this resulted in a choledochotomy rate of 23.9 per cent for the total series. Common duct stones were found and removed from 12.4 per cent of the total number of patients. There was an increase in the incidence of choledochotomy as well as an increase in the number of common duct stones found in the series of elderly patients.

There were 3 deaths in the entire series and all occurred in older patients. This comprised a mortality rate of 2.4 per cent in patients 60 years of age and older in comparison with no mortality in younger patients. The mortality of the total series was 0.7 per cent. An abstract of the 3 deaths is presented.

The use of antibiotics seemingly had little effect on the complication rate and hospital course of patients in this series and it is urged that antibiotics be used on specific indications only and not as routine accompaniment of biliary tract surgery.

A comparison of the 2 series of patients in different age groups revealed that

the older patients showed an increased incidence of jaundice, of unrelated pathologic processes, of impaired liver function, of acute cholecystitis, of carcinoma, of choledocholithiasis and an increased complication and mortality rate.

It is urged that every effort be made to operate upon patients with cholecystitis before the occurrence of complications, because along with complications comes increase in the mortality rate.

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SIMULTANEOUS SMALL AND LARGE BOWEL OBSTRUCTION RESULTING FROM CHRONIC REGIONAL ENTERITIS

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The chronic phase of regional enteritis is, in most instances, progressive in character and, in a high percentage of cases, surgery becomes necessary to control the ensuing complications of obstruction, perforation, abcess or fistula formation.

It appears somewhat interesting and unique in one's clinical experience to encounter a patient with simultaneous small and large bowel obstruction resulting from chronic regional enteritis. This experience takes on an added uniqueness when 2 such patients were observed during a relatively short interval of 4 months.

The purpose of this report is to present in some detail the clinical features of these 2 cases—the radiographic findings and methods of management with follow-up results.

CASE REPORTS

Case 1. A 23 year old man was admitted to the hospital on April 1, 1954 with a complaint of recurrent cramping lower abdominal pain of approximately 8 years' duration. There had been no melena or tarry stools. Five years prior, a gastrointestinal series and barium enema done elsewhere were reported as negative. During the past several years there has been a 65 pound loss in weight. Three weeks before admission, the cramping abdominal pain became more severe, was associated with nausea and vomiting and, at this time, pain in the left lower quadrant was noted, with some difficulty passing gas and increasing difficulty with bowel movements. There was a 15 pound weight loss during the 3 weeks prior to admission along with marked anorexia, weakness and lassitude.

Past history revealed that the patient had undergone 5 operations for drainage of perirectal abscesses during the past 2 years. There was no history of tuberculosis, diabetes or other constitutional disease. Family history revealed no evidence of regional enteritis, ulcerative colitis or gastrointestinal tract neoplasm.

On examination the abdomen was distended, particularly below the umbilicus, and a tender mass was present in the left lower quadrant of the abdomen which extended over to the midline. The liver and spleen were not palpable. Peristalsis was hyperactive on auscultation. Rectal examination revealed considerable scarring about the perianal region as a result of previous surgery but the sphincter tone was good. No rectal masses were noted.

Laboratory Studies: The hemoglobin was 12.9 Gm. per 100 cc, white blood cells were 11,800 per cu. mm. with 95 per cent segs, red blood cells were 4.9 million per cu. mm. and PCV 38. Urinalysis was normal. Serology was negative. NPN was 28 mg. per cent and fasting blood sugar 110 mg. per cent. TSP was 5.5 with ratio of 1/.75. Serum bilirubin was 0.4 mg. Prothrombin time was normal. Chlorides were 98 meq/l, CO: 22 meq/l, blood calcium 10.6 and phosphorus 3.5. Repeat stool examinations were negative for occult blood, ova and parasites. Bleeding and clotting time was normal. Cephalin flocculation was 0 after 48 hours. The Frei and tuberculin tests were negative. Sigmoidoscopic examination was normal up to the 17 cm. level. A flat film of the abdomen revealed a rather large amount of gas throughout the small bowel and a moderate amount of gas in the colon. This picture was compatible with intestinal obstruction. Barium enema revealed a constricting defect

From the Edwards-Eve Clinic, Nashville, Tennessee.

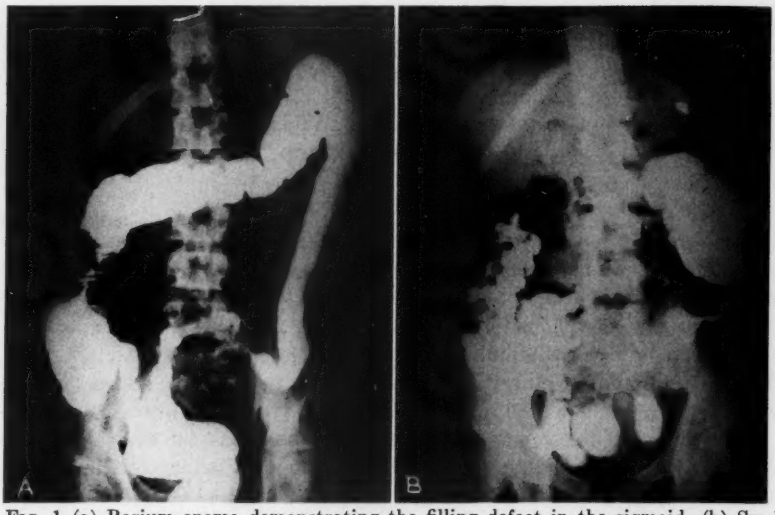

FIG. 1 (a) Barium enema demonstrating the filling defect in the sigmoid. (b) Small bowel series demonstrating the dilated and constricting segments of small bowel.

in the midsigmoid colon measuring approximately 5 cm. which the radiologist believed possibly represented an extraluminal mass producing pressure on the sigmoid, as the colonic mucosal pattern was not abnormal. After decompression with a Miller-Abbott tube, a small bowel series revealed several constricting areas throughout the small bowel and, in other areas, segments of the small bowel appeared dilated (fig. 1). After 20 hours, there was residual barium in the small bowel. Chest film was negative.

Operation: The abdomen was explored under spinal anesthesia and, upon opening the peritoneal cavity, a huge mass of small intestine was encountered occupying practically the entire left portion of the abdomen and extending across the midline. This mass showed the typical distinguishing characteristics of chronic regional enteritis and was, in turn, adherent to the arch of the sigmoid colon. The diseased small bowel was easily separated from the sigmoid and there appeared to be no fistulous tract connecting the two structures. A very extensive resection was carried out, taking care to remove a generous segment of normal-appearing small intestine on either side of the obviously diseased segment. Fortunately, the terminal ileum was not involved in the process and the ileocecal valve was left intact. An end to end anastomosis was carried out and it was estimated that approximately 2½ feet of small bowel remained (fig. 2). The segment of sigmoid colon involved in the perisigmoidal inflammatory mass was excised and an end to end anastomosis performed.

Postoperative Course: Postoperative course was entirely uneventful except for a diarrhea which lasted for approximately 2 weeks. This cleared up entirely and the stools became soft with the patient having 3 or 4 bowel movements daily. He was maintained on a high protein, high fat, and low carbohydrate diet. Blood chemistry studies, including calcium determination, remained normal and glucose tolerance curve was not remarkable.

Pathology Report: The specimen showed the typical gross and microscopic findings characteristic of chronic regional enteritis and it was interesting to note that the bowel on either side of the gross lesion, for a considerable distance, showed evidence of early mucosal and submucosal involvement. However, on each transected end, the bowel showed no microscopic evidence of disease. The colonic specimen showed only an inflammatory reaction involving the outer coats of the bowel wall.

FIG. 2 Postoperative gastrointestinal series showing the short length of bowel remaining

Follow-Up: During a two year follow-up the patient has had no further digestive disturbances, has gained 50 pounds in weight, and has 2 or 3 formed stools daily. No anemia has resulted.

Case 2. This 24 year old man was admitted to the hospital on Aug. 26, 1954 with generalized abdominal pain associated with nausea and vomiting of several days' duration. Pain was described as cramping in character, more marked in the right lower quadrant, but also was associated with left lower quadrant pain and increased difficulty in having a bowel movement.

Past history revealed the presence of vague abdominal discomfort for the past 9 months with the patient undergoing an appendectomy elsewhere 7 months prior with no relief of symptoms. There had been a 30 pound weight loss the past several months associated with malaise and anorexia.

On examination there was a large mass occupying the entire right lower portion of the abdomen which extended across the midline which did not move with respirations. The liver and spleen were not palpable. Rectal and sigmoidoscopic examinations were normal.

Laboratory Studies: Hemoglobin was 14.9 Gm. per 100 cc., hematocrit 42 and red blood cells were 5.2 million. White blood cells were 11,250 with 63 segs. Serology was negative and urine was normal. Examination of stools for ova, blood and parasites was negative. Barium enema revealed a defect in the lower sigmoid colon which was thought to represent extrinsic pressure on the colon as the mucosal pattern apparently was normal. The medial wall of the cecum also showed an extrinsic pressure defect. A Miller-Abbott tube was passed and,

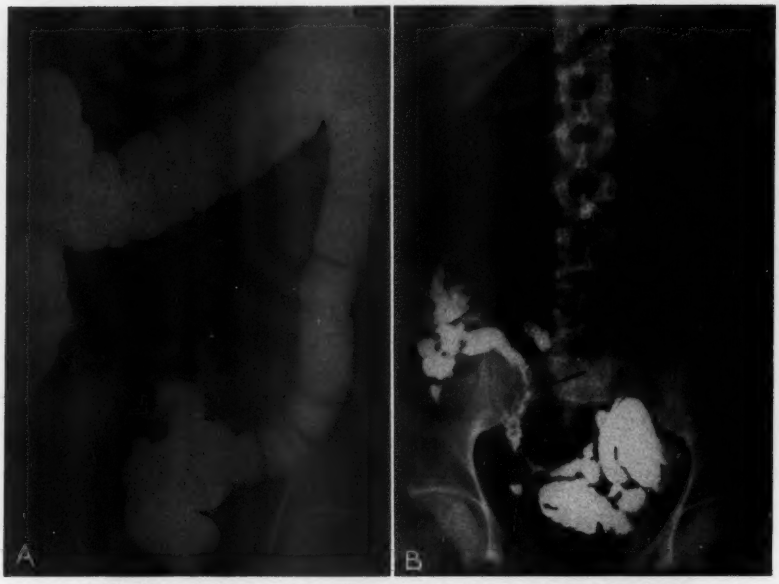

FIG. 3 (a) Barium enema demonstrating the defect in the sigmoid and pressure defect on the medial wall of the cecum. (b) Diseased segment of terminal ileum demonstrated by barium study.

after adequate decompression of the small bowel, a progressive small bowel series showed a hyperplastic mucosal lesion in the terminal ileum (fig. 3). Roentgenogram of the chest was negative. The preoperative impression was possible chronic regional enteritis, although tuberculosis and lymphosarcoma were considered.

Operation: At operation a large inflammatory mass occupying the distal small intestine, cecum and ascending colon was noted. This mass was in turn adherent to the lower sigmoid colon. The lesion had the typical gross characteristics of regional enteritis. The small bowel was separated from the sigmoid colon and no fistulous connection between the two structures was noted. A resection of the distal 40 cm. of the ileum and entire right colon was accomplished and an end to end ileotransverse colostomy performed. The inflammatory mass in the lower perisigmoidal region was resected and an end to end anastomosis carried out. Postoperative course was entirely uneventful.

Pathology Report: The gross and microscopic findings involving the resected ileum were typical of regional enteritis and the disease process had spread into the cecum and ascending colon. The segment of sigmoid showed an inflammatory reaction involving the serosa and muscular coat of the bowel but the mucosa and submucosa were uninvolved.

Follow-Up: After almost 2 years, the patient has had no further trouble referrable to the intestinal tract and has gained 25 pounds in weight.

DISCUSSION

Both of these cases are of interest in that each presented clinical symptoms suggesting partial small and large intestinal obstruction which were confirmed by radiographic study.

Case one exemplifies the value of performing an extensive resection when one is

confronted with this disease as mucosal and submucosal involvement may exist for a considerable distance beyond the gross confines of the diseased segment. Failure to appreciate this fact, no doubt, explains some of the recurrences which follow a more conservative type resection. The absence of a persistent diarrhea in the case under discussion may, in part, be attributed to the preservation of the ileocecal valve and to the high protein, high fat and low carbohydrate diet followed during the postoperative period. Compensatory changes in the mucosal villi of the remaining intestine producing an increase in the absorptive area of the bowel are likewise of importance.[1]

The inflammatory reaction noted about the sigmoid colon in both cases was secondary to the disease process involving the small bowel and did not represent a distinct skip-area of the primary disease. It might possibly be argued that the inflammatory process in the perisigmoidal tissues would have resolved following excision of the diseased small intestine, but on the other hand, a permanent colonic stricture may have persisted requiring further surgery.

SUMMARY

Two cases of chronic regional enteritis producing symptoms of simultaneous small and large bowel obstruction have been presented.

The diagnostic features and methods of management have been discussed.

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ACUTE GASTRIC DILATATION

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Acute gastric dilatation is a disease that has elicited periodic interest in the literature since the eighteen hundred and seventies, but little more information is available today than at that time. We present 6 cases which have been observed at Hines Veterans Administration Hospital since 1947 and discuss salient points in the recognition and therapy of this disease. These 6 cases do not represent the true incidence of the disease at this hospital as many cases have been seen and the patients treated by gastric suction. However, the diagnosis is not carried in the records. These 6 cases are the most severe seen and of our 6 cases, 4 of them were diagnosed at the time of autopsy or surgery and 2 were diagnosed on a clinical basis and recovered.

CASE REPORTS

Case 1. C.F., a 25 year old white man, was admitted to the hospital because of a fracture of the right femur and gangrene of the right leg. Physical and laboratory examinations were noncontributory for other disease. An above the knee amputation was performed. The postoperative course was uneventful until the third postoperative day when he began to vomit. Vomiting continued for 48 hours, and when he was observed at that time there was neither abdominal pain nor tenderness. The abdomen was markedly distended and tympanitic. A Levin tube was passed with complete relief of the distention. The patient was placed on intravenous fluids and started on orange juice and broth at the end of 48 hours. Forty-eight hours later, the tube was removed and the patient's subsequent convalescence was uneventful.

Comment: Usually vomiting is thought to be a late manifestation of acute gastric dilatation. However, in this case whether it preceded or was the cause of the vomiting that occurred beginning with the third postoperative day is not certain. It would seem that if the vomiting occurred only after the acute gastric dilatation had developed, this case did not follow the usual rapid fatal course.

Case 2. M.C. a 53 year old white man, was admitted to the hospital with a fracture of the right radius and ulna. The fracture was reduced under local anesthesia. The hospital course was uneventful until 6 days later when a distended abdomen was noted but it subsided spontaneously. Three days later, the patient was told that he was going to be discharged and a few hours later his abdomen was noted to be markedly distended. However, this also subsided spontaneously. The abdominal distention recurred on the next day and he was seen by the Surgical Department in consultation. There was noted at this time marked tympany in the epigastrium and gross abdominal distention. Roentgenograms were ordered which showed an acute gastric dilatation (fig. 1). The patient was treated by suction, sedation, intravenous fluids for 48 hours and made an uneventful recovery.

Comment: Apparently the psychic trauma of being told that he was to be discharged from the hospital was enough to precipitate the acute gastric dilatation in this case.

Case 3. This 61 year old white man was admitted to the hospital because of roentgenologic evidence of pulmonary tuberculosis. Shortly after admission he began complaining of

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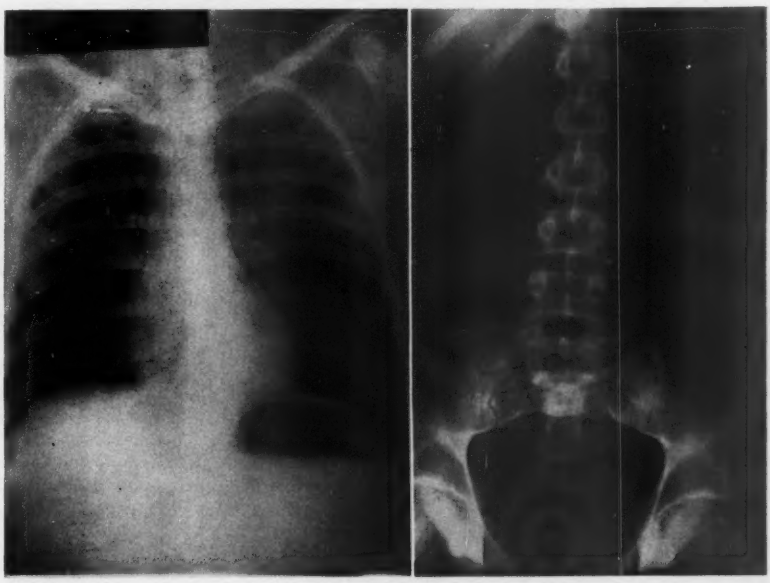
dyspnea and died 48 hours later. At postmortem examination the stomach was found to be tremendously dilated and filled the entire abdominal cavity. The small intestine was lying in the pelvis. The duodenum was found to be markedly distended (fig. 2). Pulmonary tuberculosis was also found.

Comment: This case demonstrates the speed with which acute gastric dilatation can be fatal and with only minimal symptomatology. This patient had no evidence of abdominal distention at the time of admission. His only complaint was dyspnea and he was dead within 48 hours of admission—the condition not being recognized at any time.

Case 4. This 50 year old white man was admitted to the hospital because of marked malnutrition and intermittent vomiting of 2 months duration. No significant physical or laboratory findings were present on admission. Following admission to the hospital, he continued to vomit daily. He received intravenous fluids containing dextrose and sodium chloride. Eleven days after admission he was thought to have a marked ascites. Six days later, with no relief from the vomiting or ascites, the patient died. Autopsy examination revealed the entire abdominal cavity to be occupied by a dilated stomach and duodenum (fig. 3). The stomach contained 4700 cc. of dark colored fluid. A hard mass was found in the antrum of the stomach which proved to be adenocarcinoma of the stomach. However, the pyloric ring admitted one finger with ease.

Comment: This represents another case in which the vomiting apparently preceded the acute gastric dilatation, rather than being a result of it. This case demonstrates one of the more common errors in the diagnosis of acute gastric dilatation, for there was so much fluid present in the dilated stomach that this patient was thought to have ascites. There was a carcinoma of the stomach, but it in no way obstructed the pylorus or antrum and the duodenum was markedly dilated.

Case 5. This 39 year old white man was admitted to the hospital with the complaint of a



a b

FIG. 1 (a & b). Roentgenograms showing acute gastric dilatation with the typical fluid level and air bubble in the stomach (a). This stresses the importance of an upright film for there is no evidence of this in a scout film of the abdomen in the supine position (b).

mass in the right neck of 6 months duration. A needle biopsy revealed this to be a bronchogenic carcinoma, small cell type. Several days later, the patient fell out of bed. No abdominal abnormality was noted when he was examined. However, 4 hours later he had an emesis of about 300 cc. of coffee-ground material and a fluid splash was heard in the abdomen. Approximately 14 hours after the first emesis the abdomen was found to be rigid and distended. At this time he regurgitated more coffee-ground material and very shortly thereafter died. Examination of the body revealed a hugely dilated stomach and dilated duodenum down to the ligament of Treitz (fig. 4). The stomach contained approximately 1000 cc. of dark fluid and much gas. The stomach mucosa showed many small superficial erosions. No other abnormalities of the abdomen were noted.

Case 6. This 37 year old Negro man arrived at the hospital by ambulance in a state of shock. No blood pressure or peripheral pulse was obtainable. He was treated for shock by means of fluids and vasopressor agents and at that time he stated that he had had recurrent episodes of right lower quadrant and periumbilical pain for one year. He claimed that he had had pencil-like stools for 3 days. Acute periumbilical pain of cramping nature had started 24 hours prior to admission and shortly thereafter he started vomiting, usually small amounts of bile stained material. He had been previously treated for tabes doraslis and syphilitic endoarteritis. Physical examination revealed an acutely and chronically ill patient who was somewhat dyspneic. The abdomen was markedly distended and tympanitic.

FIG. 2 Gross specimen at time of autopsy shows the marked gastric dilatation with no evidence of an obstructing lesion.

FIG. 3 This demonstrates the stomach occupying the entire abdominal cavity thus making it easy to understand how the fluid in the stomach could be mistaken for ascitic fluid.

Liver dullness was absent and there was marked rebound tenderness and abdominal rigidity. Examination of the genitalia revealed a partial priapism and marked engorgement of the superficial veins. Rectal examination was negative except for prolapsed hemorrhoids. The laboratory findings were within normal limits. A Levin tube was inserted and 200 cc. of dark fluid and several hundred cc. of air returned. Roentgenograms were then taken but it was not noted that the Levin tube was in the esophagus (fig. 5). The patient was taken to surgery with a preoperative diagnosis of ruptured viscus. Under local anesthesia, the abdomen was opened and an acute dilatation of the stomach was recognized. An attempt was made to decompress the stomach slowly by means of a no. 20 needle, but within a few minutes after decompression was started, cardiac arrest occurred. Although immediate resuscitative measures were instituted he failed to respond. Operative examination of the abdomen revealed the small bowel to be of normal size distal to the ligament of Treitz. No abnormalities of the stomach or duodenum other than marked dilatation were noted. The large bowel was not examined.

Comment: Because no autopsy was performed, the underlying disease in this case is unknown. It would seem that death in this instance might not be related to acute gastric dilatation per se but rather to the sudden changes in circulation and right heart failure that could occur with decompression.

DISCUSSION

Although most reported cases of acute gastric dilatation occur following operations, it may occur as a complication of nearly any physical or mental trauma. It

has been seen following the application of a cast, hence its name, "Cast Syndrome"[3]. It most frequently is seen after relatively minor abdominal operations; interestingly enough it is practically never seen after gastric or duodenal surgery. Probably this is due to the routine use of a nasogastric tube in gastric surgery. Acute gastric dilatation has been known to occur in some people with no other basis than fear or psychic trauma. Gastric distention by means of positive pressure anesthesia without an endotracheal tube may also result in acute gastric dilatation. This has been pointed out by Maddock[7, 8] who showed that even more gas is forced into the stomach if curare or curare-like drugs are used. In this instance it is easier to explain the cause of acute gastric dilatation, yet the end results may be the same as in any other case no matter how it is initiated.

Even today little is known about the true mechanism of acute gastric dilatation. That this condition was caused by an obstruction to the third portion of the duodenum by the mesenteric vessels was disproved by the Dragstedts[4] in 1922. They showed that when complete obstruction of the third portion of the duodenum resulted in death there was no concomitant dilatation of the stomach and duodenum. Dragstedt and associates[5] in 1931 further developed Brinton's[2] atonic

FIG. 4 This autopsy specimen reveals the marked gastric dilatation that can occur in a short period of time. This is known to have developed within 18 hours.

a b

FIG. 5 (a & b). These roentgenograms fail to reveal a fluid level in the stomach because of the positioning of the patient at the time they were taken. However, they do reveal the dilatation of the stomach. It is so marked that there appears to be free air under the diaphragm (a). In (b) the semicircular line of increased density seen overlying the pelvis is the greater curvature of the stomach.

theory by proposing that it was due to a reflex inhibition of the peripheral gastric motor mechanism through efferent impulses reaching the stomach by way of the vagi and the splanchnics. He thought the stimulation of any visceral or somatic sensory nerve could produce reflex inhibition. Still another mechanism was advanced by Morris, Ivy and Maddock[9] in 1947 when they suggested that air sucking was the method involved. They thought that nearly everyone would suck air into the stomach with their respiratory movements if it were not for the resistance of the normal superior esophageal sphincter. However, it was their belief that this sphincter might be relaxed by general debilitation, anesthesia, or some unexplained reflex and thus initiate acute gastric dilatation. Starr[10] has recently suggested an entirely different theory. He believes that a disturbance of the electrolyte circulation of the intestine produces certain adynamic states which are related to the nature of the original disease. Thus, ileus of the proximal intestine, identical with acute gastric dilatation, is caused by a disproportion in potassium and chloride loss. On the other hand, he believes that ileus of the distal intestine, identical with the usual paralytic ileus, is caused by a disproportion of sodium and chloride loss. This theory might be adequate in some of our patients who lost

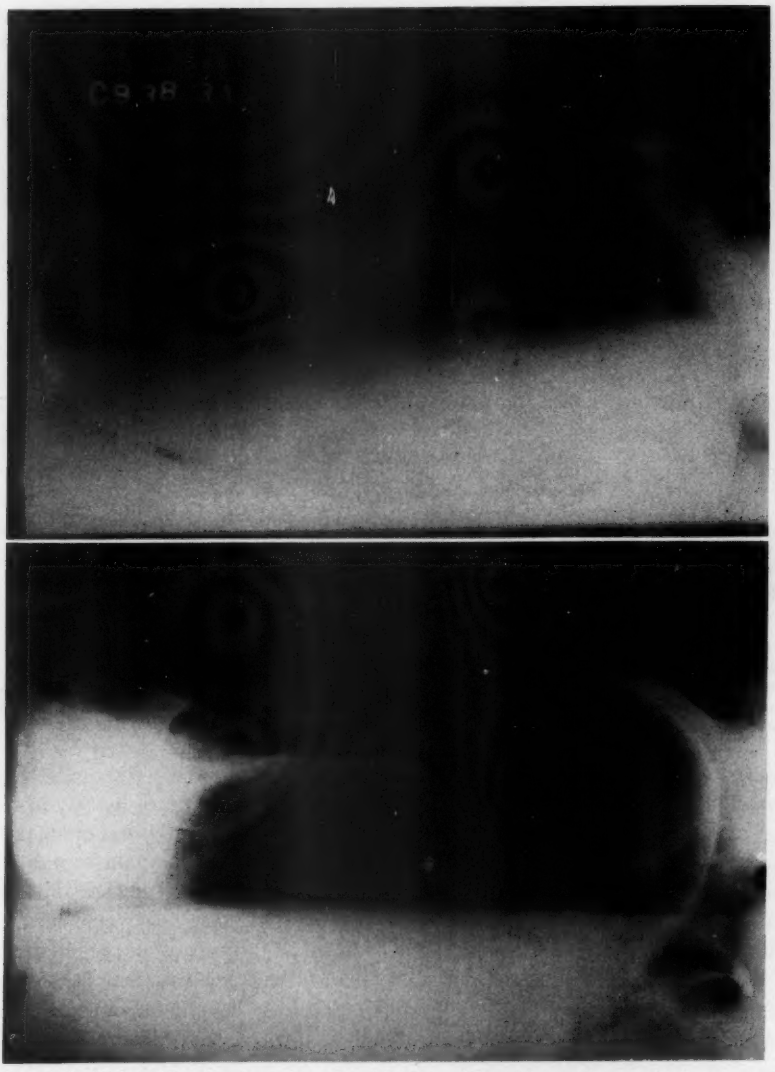


FIG. 6 (a & b). (a) Supine decubitus (b) right lateral decubitus, adequately demonstrate an air fluid level in the stomach when viewed in conjunction with the films in figure 5 (a & b). This is a most important roentgenologic finding.

potassium but it would be difficult to explain the acute gastric dilatation in the patient who did not want to leave the hospital, Case 2.

Regardless of the initiating mechanism of acute gastric dilatation, there are three events in the progression of the disease that complicate treatment. The

first event was pointed out by Dragstedt and associates[5]. As the dilatation of the stomach progresses, it pushes the small bowel into the pelvis and in so doing drags the superior mesenteric vessels tightly over the third portion of the duodenum and actually causes a mechanical obstruction at that point. The second event complicating this disease is the marked angulation which occurs at the esophageal hiatus, as it is a fixed point in the body and the stomach distends lateral to this point. This angulation may make it difficult, if not impossible to pass a nasogastric tube. The third complicating factor is that most people become aerophagics under conditions of stress and the swallowing of air can occur even when the angulation of the cardia is very severe. This causes the acute gastric dilatation to progress even more rapidly.

Clinically, acute gastric dilatation is not difficult to recognize if it is considered in the differential diagnosis. Proper roentgenograms will confirm the diagnosis. Demonstration of a fluid level in the stomach is most important and this can be done by means of the upright film of the abdomen or lateral decubiti films (figs. 1a and 6). The best therapy for acute gastric dilatation is prevention. As has been pointed out by others[1, 6], intubation of the stomach should be done at the slightest suspicion of its presence. The further the disease progresses, the harder it is to institute proper treatment.

Sometimes it is possible to pass a large tube such as an Ewald tube through the area of acute angulation at the esophago-cardiac junction. Fluid and electrolyte replacement must be adequate and Starr[10] suggests the potassium replacement must be massive. This is certainly suggested in several of our cases when death occurred from acute gastric dilatation and no potassium had been given. Surgical therapy is not indicated in the early stages of the disease and it is, at best, very dangerous but necessary in the late stages. The only aim of surgical therapy in this disease is decompression of the stomach to the point that a nasogastric tube can be passed into the stomach. However, the most crucial part of surgical therapy is the very necessary, slow decompression of the stomach after opening the abdominal wall so that the body can accommodate to the changes in pressures that take place. Even slow decompression may be too rapid as demonstrated in Case 6.

SUMMARY

Even with modern methods of treatment, acute gastric dilatation can be an acute and rapid cause of death.

Mortality in unrecognized and untreated cases is extremely high.

Intubation of the stomach must be done at the slightest suspicion of its presence.

Electrolyte and fluid therapy must be adequate.

Surgical decompression is, at its best, a very dangerous procedure.

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TREATMENT OF PERIPHERAL ARTERIOSCLEROTIC DISEASE BY ARTERIAL HOMOGRAFTING

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It has long been recognized that arteriosclerotic narrowing, occlusion, or dilatation of peripheral arteries can be part of an extensive generalized process or can be segmental in nature. It was not until arterial grafting became generally possible that the therapeutic significance of the segmental type of disease was fully apparent. Before the advent of grafting, therapy included general support, care of the feet, and at best the use of such operative procedures as lumbar sympathectomy or endarterectomy. It is now possible, in many instances, actually to restore main vessel blood flow and achieve results previously unattainable.[4]

Much has been written about disease of the thoracic and abdominal aorta[2] but since sclerosis of the vessels distal to the aorta is much more common and presents a somewhat different problem, we are presenting here our experience with replacement of 24 of these distal vessels. Four of the grafts appear to have become occluded, and an attempt is made to analyze the cause of these failures. We have used, in all cases, arterial homografts, either frozen or freeze-dried[1], from the Baylor Hospital blood vessel bank maintained in the Wadley Blood Center. There have been no deaths in this series.

ILIAC ARTERY DISEASE

The symptoms produced by decreased flow through an iliac artery are dependent on the rate of development of the obstruction, the amount of collateral flow, and the condition of the arteries distal to the point of narrowing. In some cases the only complaint is claudication in the gluteal, thigh, or leg muscles. The appearance of the limb may be normal. Such cases are the best candidates for arterial homografting. Patients with peripheral ischemia who exhibit night pain and tropic changes in the skin are more likely to have poor distal vessels, and unless the occlusion has occurred suddenly, are less likely to obtain benefit from a homograft. A clinical diagnosis of iliac artery insufficiency is made by finding an absent pulse below the inguinal ligament on one or both sides with a palpable aortic pulse. This diagnosis is confirmed by aortography. In cases of iliac occlusion, the needle usually is inserted at the upper margin of L 3, and 30 cc. of 70 per cent Urokon injected. It occasionally is necessary to do femoral arteriograms in addition to determine the patency of the femoral and popliteal arteries.

Eleven iliac arteries have been replaced in 9 patients. Two patients have had bilateral iliac involvement and in these it has been technically easier to replace the distal aorta and both common iliac arteries than do separate iliac grafts. The

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follow-up time in these has ranged from 14 months to 3 weeks. Ten of the 11 limbs have satisfactory blood flow and are free of symptoms at this time. One of these 10 was found to have a nonpulsating graft when examined 8 months after operation, although clinically the leg is much improved and is symptom free. In one patient it was recognized at the time of operation that the distal vessels were not ideal, but a by-pass type graft was inserted. Aortography 3½ months after operation revealed that it was completely occluded. Ischemia of the foot had progressed so that it was necessary to amputate the leg because of gangrene.

The first patient in whom a lumbar sympathectomy was not performed at the time of homografting was one with bilateral iliac artery involvement. This patient had complete obstruction of the right common iliac artery and marked narrowing of the left. He had previously had a nucleus pulposus removed for right hip pain, without improvement. He had no symptoms on the left side. When he recovered from anesthesia, he complained of severe pain in the toes of the previously asymptomatic left foot. Dorsalis pedis and posterior tibial pulses were palpable but the toes were cold and blue. Four toes became gangrenous and had to be amputated, although the circulation of the remainder of the limb remained excellent, and the limb is asymptomatic at this time.

FEMORAL ARTERY DISEASE

Femoral arterial occlusion has long been recognized as one of the commonest diseases producing symptoms in the lower extremities. Although claudication alone may be present it is more likely to be accompanied by signs of ischemia of the skin and nerves. The pulsation of the common femoral artery can be felt but there is no pulsation of the popliteal artery or of the arteries of the foot. Oscillometric studies may reveal excellent indices in the upper thigh with either weak or absent pulsations near the knee and in the calf. The demonstration of segmental occlusion is made by percutaneous injection of 20 cc. of 30 per cent Urokon into the femoral artery just below the inguinal ligament. Many patients have been found to have patent femoral vessels but with marked narrowing and very small popliteal arteries, without any point of complete obstruction. These have not been thought suitable for grafting.

Eight femoral arteries in 7 patients have been found to have segmental occlusions with patent popliteals. By-pass grafts of the end to side type with lumbar sympathectomies have been done in these cases. Six limbs are viable. Two of the grafts have occluded. One patient had a very narrow popliteal artery distal to the point of obstruction, and poor retrograde flow was obtained at the time of operation. The graft in this limb appeared to clot immediately after operation. However, the lumbar sympathectomy appears to have been sufficient to allow healing of the spots of gangerene on the toes, and his only symptom at this time is claudication in the calf muscles. Another patient with a previous lumbar sympathectomy had lost two toes with metatarsal heads because of gangerene. An open wound persisted. An arterial graft was inserted and it has remained patent. However, an injury of the foot 3½ months after the insertion of the graft resulted in infection and further gangerene, and a leg amputation was necessary. A third

patient, with a palpably patent graft, received an undue exposure of his foot to extreme cold, which resulted in gangerene of the great toe and foot, necessitating leg amputation 8 months after grafting.

One patient exemplifies the numerous types of complications which may arise in those who have generalized arteriosclerosis, even though segmental occlusions also are present. This man had segmental occlusions of both femoral arteries and on admission had gangerene of the tip of the right great toe. Lumbar sympathectomy and the insertion of a 6 cm. by-pass graft resulted in prompt healing of the toe and disappearance of all symptoms. On the eighth postoperative day he developed thrombophlebitis of the femoral vein with massive swelling of the foot, leg, and thigh. His pedal pulses, which had been present, were not palpable through the edema of the foot, and when the edema subsided it was obvious that the graft had become clotted. However, the period of increased arterial supply, with the lumbar sympathectomy, was sufficient to leave this foot and leg asymptomatic. Two months later a lumbar sympathectomy and a 40 cm. end-to-side graft from common femoral to popliteal artery was done on the left. This caused prompt disappearance of his previously existing calf claudication. Five weeks after the last procedure he suffered a myocardial infarction. At the end of 3 weeks he was allowed to stand for the first time and at that point there was a sudden embolization of the left common femoral artery. He was already receiving Heparin, and within an hour was started on Heparin. He was quite ill and was treated at his home, in a distant city with anticoagulants and autonomic blocking agents. Ischemia of the foot progressed and by the twenty-seventh day after the embolus blebs were forming on the foot, which was quite painful, although his general condition was improved. At that time he was transferred to Dallas and his common femoral artery was explored. It was possible to remove the embolus. The profunda femoris was found to be clotted for only 2 cm. and excellent flow was obtained after removing the clot. The graft was filled with clot, which could be readily expressed from below upward, and back flow of blood resulted. The embolectomy resulted in immediate healing of all lesions of the foot, and the limb has now become asymptomatic. The graft itself has a palpable pulsation. His activities are still being restricted because of heart disease.

POPLITEAL ARTERY DISEASE

Involvement of the popliteal arteries by arteriosclerosis may be manifested by narrowing, with or without occlusion, or by aneurysmal dilatation. The symptoms of popliteal artery occlusion are similar to those of femoral artery occlusion; namely, calf claudication with or without signs of skin and nerve ischemia, such as pain and gangerene. The diagnosis is made by the finding of a femoral pulse with absent popliteal and pedal pulses. It is confirmed by femoral arteriography. These occlusions are not suitable for homografting, since the bifurcation usually is involved and the anterior and posterior tibial arteries usually are too small to accept grafts.

Arteriosclerotic aneurysms of the popliteal arteries lend themselves especially well to homografting. It is imperative that these lesions be treated as soon as

possible after their discovery because their prognosis is very poor if thrombosis occurs.[3] The symptoms of the aneurysm before thrombosis vary from none to pain in the foot and leg. The finding is that of a pulsating mass in the popliteal fossa. In questionable cases femoral arteriogram will confirm the diagnosis. When thrombosis occurs, there usually is the sudden onset of pain in the foot and leg with disappearance of the pedal pulses. A firm mass can be felt in the popliteal fossa. Lumbar sympathectomy with excision of the aneurysm has been the accepted treatment and usually will result in the preservation of the foot and leg. However, claudication with other signs of ischemia commonly follows this procedure. We believe the combination of lumbar sympathectomy, excision of the aneurysm, and homograft replacement is the treatment of choice for these lesions.

Five popliteal aneurysms have been resected and replaced by homografts. Lumbar sympathectomy has been performed in each case. All five grafts have been successful and the circulation of the limbs is near normal. When one patient was first seen he had obvious ischemia of one foot and leg and a thrombosed popliteal aneurysm was found to be present. A pulsating popliteal aneurysm was present in the other limb. The clotted aneurysm was resected and a lumber sympathectomy done. The foot survived but rather marked claudication persisted. The contralateral aneurysm was electively resected and replaced by homograft and a lumbar sympathectomy done. There are no symptoms in this limb.

Another patient, a 68 year old white man presented himself with pain in one leg. Upon examination bilateral popliteal aneurysms were found. Homografting with sympathectomy was urged but he declined to have any treatment. He returned 11 months later, complaining that 3 hours earlier the right leg had become cold, white, and numb. The aneurysm no longer pulsated. Six hours after the onset of symptoms a lumbar sympathectomy was done and the aneurysm replaced by a homograft. Ecchymotic spots on the toes and intradermal bleeding on the foot were already present. The foot was swollen and the calf was tender for several days, but at the end of 2 weeks no symptoms remained. One week after the first operation, elective sympathectomy and homograft replacement of the popliteal aneurysm in the left leg was performed. This leg is asymptomatic.

DISCUSSION

Arterial homografting represents the ideal treatment for arteriosclerotic lesions of the peripheral arteries, provided certain requirements are met. There must be adequate proximal blood flow, and the distal vessels must have the lumina to accept a sufficient volume of blood to maintain a good rate of flow or the graft will become occluded with clot. The three grafts which became clotted due to poor distal vasculature were inserted early in this series, and have taught us to be extremely critical of the recipient vessels beyond the point of obstruction. However, an occasional by-pass graft is still inserted in which the distal vasculature is questionable, when this can be done without interfering with any of the collateral flow. Although it is obvious that careful selection of the arterial

homografts, meticulous technic in their insertion, and maintenance of adequate blood pressure and blood volume are necessary for the success of these grafts, probably no other single factor is so important as having an arterial bed distal to the graft which can receive the blood brought to it by the graft. Two other conditions which may result in the loss of function of a graft are adjacent thrombophlebitis and embolization into the origin of the graft. It must be constantly borne in mind that most of these patients have generalized arteriosclerosis, as well as segmental occlusions, and the patient as a whole must be constantly watched.

End-to-end anastomoses have been used in iliac and popliteal artery grafts. End-to-side anastomoses have been used in the femoral grafts and at the distal end of the iliac artery grafts when the vessels have been carried into the thighs and connected to the femoral arteries. Distal heparinization should be done while the vessel is clamped in order to prevent clotting of the arteries beyond the obstruction. General heparinization is not used. All clots and arterial debris must be removed from the vessels proximally and distally prior to the restoration of blood flow.

When careful selection of patients for grafting is carried out and when the conditions listed above are met, success in arterial grafting can be counted on in a high percentage of cases. Main vessel blood flow can be restored, limbs can be saved, and patients rehabilitated in a striking manner.

SUMMARY

The results of arterial homografting in 24 arteries distal to the aorta in 20 patients are presented. The results in general have been quite good. There were no deaths in the series, and some limbs with obvious early gangrene have been saved. Four grafts have become occluded. Certain pitfalls in homografting procedures have been outlined.

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NECROTIZING PANARTERITIS: A COMPLICATION OF REPAIR OF COARCTATION OF THE AORTA

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Since 1945, when Crafoord[3] and Gross[7] independently reported successful surgical repair of coarctation of the aorta, numerous large series of cases have demonstrated that operative treatment of this anomaly is quite satisfactory and relatively safe. A review of the literature for the untoward sequellae of surgery reveals that hemorrhage, dehiscence of the aortic suture line and cardiac arrest, in the order named, are the common causes of surgical mortality. Postoperative hemothorax, empyema and paraplegia are the usual conditions producing postoperative morbidity. It is our purpose to call attention to necrotizing panarteritis as a serious, occasionally fatal, postoperative complication and to suggest a method for its treatment.

In spite of the extensive literature on the surgery of coarctation, there is surprisingly little mention of necrotizing panarteritis or, even, postoperative abdominal symptoms. In reporting upon 270 operations, Gross[6] makes no mention of this syndrome. In a study of 70 patients, Claggett and Jampolis[2] report the case of one patient who evidenced severe abdominal symptoms on the ninth, tenth, and eleventh postoperative days with recovery. In reporting 22 operations, Sealy[17] recorded the case of one patient with this syndrome who developed severe abdominal pain on the sixth, and died during abdominal exploration on the tenth postoperative day. Lober and Lillehei[9] reported 2 cases of severe necrotizing panarteritis with one death. Perez-Alvarez and Oudkerk[10] had a similar patient who developed symptoms on the eighth and died on the twenty-seventh days after operation. They also mentioned 2 other patients in their series, one of whom had abdominal pain and the other transient hematuria. This review has disclosed to us only 5 published cases of this syndrome with 2 recoveries and 3 deaths. To these we would add one fatal case of necrotizing panarteritis and 6 recovered patients apparently representing less severe forms of the disease. Our experience includes 72 patients with repair of aortic coarctations.

Case 1. An 8 year old white male child was found to have a heart murmur during a routine examination. His blood pressure was 140/100 in both arms. The blood pressure in the legs was 40 mm. mercury by the blanching method. There was minimal cardiac enlargement by roentgenologic examination. On May 12, 1952 an adult type coarctation of the aorta was excised and an end to end anastomosis of the aorta accomplished. There were good femoral pulses following the operation. On the fifth day following operation he began to complain of abdominal pain. There was slight abdominal distention and mild diffuse abdominal tenderness. The temperature was 101F. and the white blood cell count was 20,000 per cu. mm. with 84 per cent polys. He continued to complain of pain and the findings on physical examination remained the same. During this period he ate and drank small amounts of food and

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fluids and had bowel movements. Increased abdominal distention and increased generalized tenderness were present on the nineteenth postoperative day and laparotomy was performed. There were hundreds of small areas of gangrene involving almost the entire small intestine. Many of the gangrenous areas had perforated resulting in localized abscesses. The intestinal wall between the areas of gangrene appeared relatively normal except for evidence of inflammatory reaction. All of the small intestine from 2 inches below the ligament of Treitz to 2 inches from the cecum was resected and an end to end anastomosis performed. The child died on the sixty-ninth hospital day. During the postoperative course there was dehiscence of the abdominal wound, marked diarrhea and progressive weight loss. Examination of the surgical specimen revealed many areas of mucosal necrosis and ulceration. The underlying submucosa was edematous, markedly congested, infiltrated with inflammatory cells, particularly perivascularly, and many ulcerations were present with early granulation tissue formation. Small areas of intact mucosa were heavily infiltrated with inflammatory cells. The muscularis was thinned, congested, inflamed and focally necrotic. All the vessels were dilated and engorged; some presented fibrinoid degeneration of the wall while large arteries presented a picture of medionecrosis. A few had thrombosed lumens. The lymph nodes had enlarged, distended sinusoids filled with monocytes, some polymorphonuclear cells and eosinophils (figs. 1 and 2). The pathologic diagnosis was necrotizing panarteritis with mesenteric thromboses.

Case 2. An 8 year old white male child complained of tingling in the legs after running and headaches for 6 months. Examination revealed a grade 3 systolic murmur over the precordium. His blood pressure was 152/94 in the right arm and 148/88 in the left arm. The blood pressure in the legs was unobtainable by the cuff method. On July 7, 1955 an adult type coarctation of the aorta was excised. The proximal end of the left subclavian artery was anastomosed end to end to the distal cut end of the aorta. Diffuse abdominal pain appeared on the third day following operation. There was mild abdominal distention and tenderness in both lower quadrants of the abdomen. A roentgenogram of the abdomen revealed small and large bowel distention. The white blood cell count was 15,800 with 86 per cent granulocytes. His temperature was 101F. A Miller-Abbot tube was passed into the

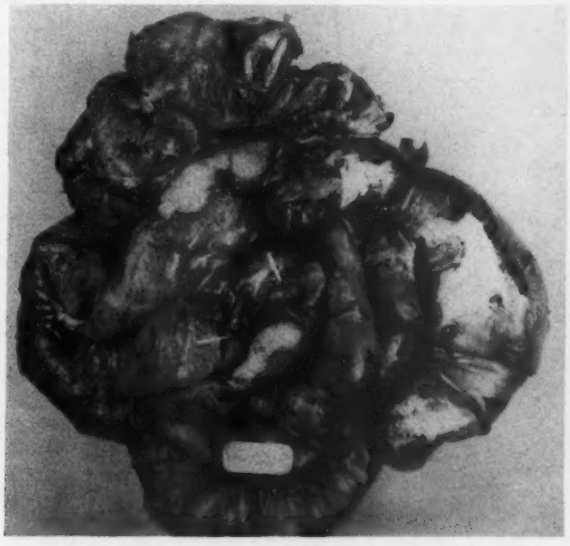

FIG. 1 Gangrenous small intestine showing multiple perforation

duodenum and allowed to advance into the small intestine. Neomycin and sulfathalidine were given orally to sterilize the bowel. He continued to complain of abdominal pain and diffuse tenderness, both direct and rebound, were present over the next several days. The bowel sounds were decreased in intensity. By the eleventh postoperative day these findings and complaints were no longer present and the Miller-Abbot tube was removed. He was dismissed from the hospital on the seventeenth postoperative day.

Case 3. A 3 year old boy had had swelling of eyes, hands, legs and body one year prior to the present hospital admission. Bulging of the precordial region also was noted at that time. Digitalis therapy was started and continued to the time of this admission. Examination revealed moderate bulging of the precordial region. There was a grade 3 systolic murmur at the base of the heart. His blood pressure in his arms was 170/80 and in his legs 90/70. Roentgenologic and fluoroscopic examination showed the heart to be greatly enlarged. There was marked pulmonary venous congestion. At operation on Feb. 11, 1956 an adult type coarctation of the aorta was excised and an end to end anastomosis performed. On the third postoperative day he complained of abdominal pain and refused food and fluids. The temperature was 101.5F. The abdomen was mildly distended and there was generalized mild abdominal tenderness. The white blood cell count was 23,900 with 89 per cent filamented cells. A Miller-Abbot tube was passed and neomycin and sulfasuxidine started. Terramycin was given intravenously. The abdominal findings gradually subsided and the tube was removed on the tenth postoperative day.

Case 4. A 6 year old white boy had excision of an adult type coarctation of the aorta and end to end anastomosis on July 18, 1950. On the third postoperative day he complained of periumbilical pain. The temperature was 101.6F. and there was abdominal tenderness, more marked in the right lower quadrant of the abdomen. There was tenderness on the right side on rectal examination. Peristalsis was decreased. White blood cell count was 22,750 with 89 per cent polymorphonuclear leucocytes. A diagnosis of acute appendicitis was made and laparotomy performed. At operation a considerable amount of thin free

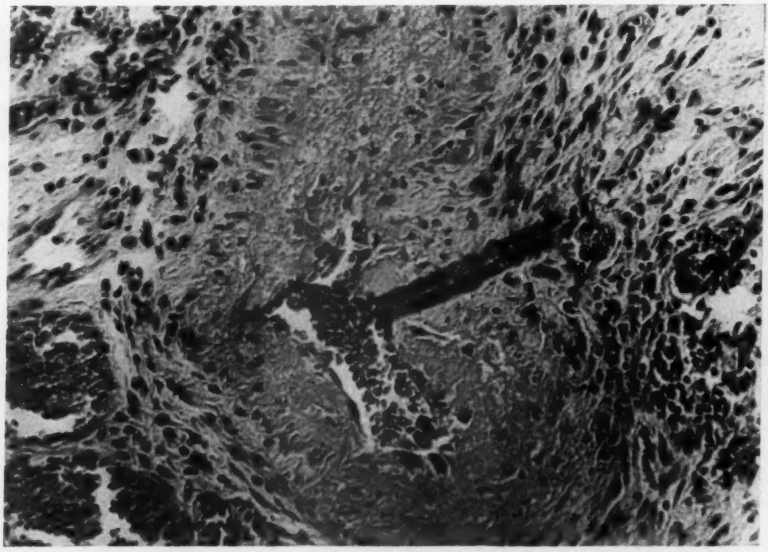

FIG. 2 Mesenteric artery showing periarterial inflammation. × 240

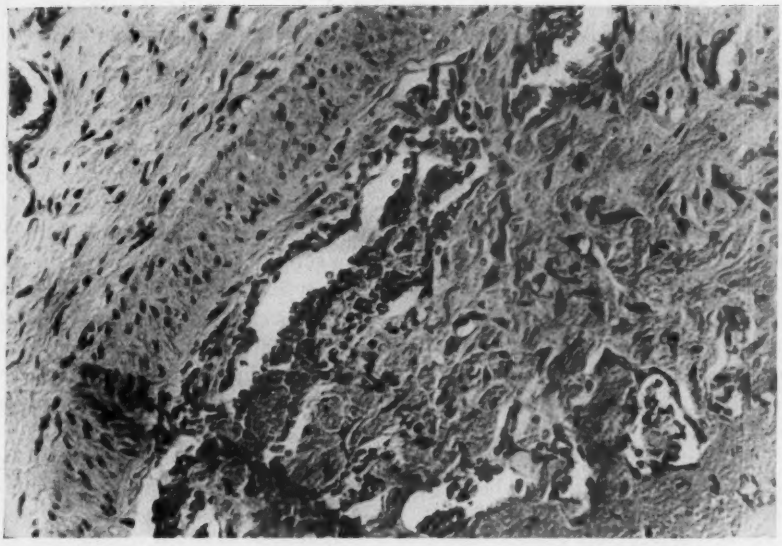

FIG. 3 Mesenteric artery containing an organizing thrombus. × 240

peritoneal fluid was found. The lymph nodes in mesentery were enlarged. At the time a diagnosis of mesenteric adenitis was made. The appendix was removed. The child continued to have abdominal distention and pain for 6 days following this operation. The microscopic sections of the appendix were reviewed but no evidence of panarteritis was found.

Case 5. A 6 year old boy underwent excision of an adult type coarctation of the aorta on March 17, 1954 with an end to end aortic anastomosis. Two days postoperatively he complained of abdominal pain. There was abdominal distention and generalized tenderness. By the sixth postoperative day the abdominal findings had disappeared and the child became asymptomatic.

Case 6. A 7½ year old white male underwent excision of an adult type coarctation of the aorta on July 29, 1952. The proximal end of the left subclavian artery was anastomosed to the distal end of the aorta. On the second postoperative day there was mild abdominal distention with diffuse abdominal tenderness, both direct and indirect. These signs disappeared by the fifth postoperative day.

Case 7. A 19 year old white female had excision of an adult type coarctation of the aorta on Jan. 22, 1949 with end to end anastomosis. Diffuse abdominal pain appeared on the third postoperative day and continued for the following 3 days without localizing. The possibility of acute appendicitis was considered but the pain and findings disappeared by the seventh postoperative day.

DISCUSSION

As in the other fatal cases that have been reported, the autopsy on our patient disclosed severe vascular changes only below the level of the coarcted segment of aorta. The muscular arteries of the mesentery showed areas of acute necrosis and inflammation. The vessel lumens contained fresh and partially organized thrombus. The intima and internal elastic lamina usually were preserved. How-

ever, the media showed severe destruction with disintegration of its fibers and mononuclear and leukocytic infiltration. Lober and Lillehei[9] thought that the vascular lesions closely resemble those seen in periarteritis nodosa.

The etiology of this condition is obscure but there is experimental evidence to indicate that medial necrosis of muscular arteries results from the sudden increase in intravascular tension and blood flow in an arterial bed accommodated to both a low blood pressure and a low volume flow. The arteries of the lower extremities being supported by the muscles are less affected than those of the abdominal viscera which are not so protected. The fact that the vascular changes are localized to the area below the coarctation supports this mechanical theory and would appear to exclude factors such as hypersensitivity or response to stress which would be expected to affect the vessels of the upper part of the body as well. The patient in our fatal case received penicillin and streptomycin in the postoperative period but there was no evidence of hypersensitivity to these drugs. Beginning on the twelfth postoperative day and continuing to his death he received corticotropin in large doses without improvement.

It seems reasonable that all grades of severity of necrotizing panarteritis may occur and that the patients who recovered without any special treatment represented the mildest form of the disease. The possibility exists, of course, that the abdominal complaints merely represented a mild postoperative paralytic ileus, but the rather definite pattern of associated fever and leukocytosis seemed to indicate a more serious process. We believe that our 2 patients in whom intestinal decompression and bowel sterilization were employed, had more extensive involvement of the arteries in the intestine as shown by a more protracted course and more definite physical signs of peritoneal irritation.

Experimental work has shown that there is a decreased mortality accompanying devascularization of segments of the bowel, closed loop obstruction, strangulated bowel and in mesenteric occlusion when certain of the antibiotics were administered to sterilize the intestinal contents.[4, 13, 14, 18] It also has been shown that revascularization of segments of intestine can occur if the bowel is kept decompressed and its contents sterile.

With the outset of abdominal pain following repair of a coarctation, it seems advisable to stop all oral alimentation, maintain fluid and electrolyte balances by the intravenous route, decompress the stomach and bowel by nongastric tube, and sterilize the intestinal contents with neomycin and mycostotin by mouth. Signs of intestinal perforation or hemorrhage from one of the involved arteries should be watched for and laparotomy carried out should either occur.

SUMMARY

Attention is called to the definite clinical entity of necrotizing panarteritis as a complication of repair of coarctation of the aorta. It probably occurs much more frequently than reports in the literature would indicate. Abdominal pain, distention, tenderness, fever, decreased peristaltic sounds and leukocytosis ac-

company the condition. We have outlined a program of treatment and presented illustrative cases.

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CLINICAL EXPERIENCES WITH PREFABRICATED NYLON ARTERY GRAFTS

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The very important demonstration of Voorhees, Jeretski and Blakemore[3]—that tubes tailored from inert textile fabrics could serve as artificial arteries—is now a medical classic. Various fibers such as Vinyon N, nylon, Dacron and Orlon have been constructed into woven, knitted or braided tubes or tailored at the operating table and used successfully in experimental animals and in human beings. The search for the ideal fabric and the ideal method of fabrication is continuing in many centers.

Two years ago a joint project was set up to combine textile knowledge and facilities with surgical laboratory and clinical testing of various means of improving fabric grafts. It was thought that a fabric prosthesis to approach the *"ideal"* should have the following characteristics:

A. Elimination of clumsy seams by construction of braided or woven tubes.

B. Chemical or physical treatment of the tube so that the cut end would hold an arterial suture securely without fraying.

C. Rendering the cloth tube unwettable to prevent blood loss, yet not impermeable to fibroblastic penetration through the fabric.

D. Production of a flexible tube that would not kink so that: (1) Grafts could cross joint surfaces (groin, popliteal space, axilla.) (2) By-pass grafts around obstructed arterial segments could be constructed using the end to side technic.

E. Prefabricated Y tubes for aortic bifurcations.

Nylon was chosen as the most desirable fiber for two reasons. First, it is the toughest and most durable of the artificial fibers both from the standpoint of tenacity and fatigue strength. (Table 1). It is for this reason that it is used in airplane and automotive tire cord. Second, much more is known about the chemistry of nylon than about the relatively newer fibers. In this regard, a very satisfactory semisolvent for nylon is well known (formic acid) whereas, no satisfactory solution is known for many of the other fabrics except for acrylic fiber (Orlon, etc.), and this semisolvent has such a critical temperature of reaction as to be impractical. Nylon absorbs slightly more water than the other fibers, is therefore, slightly more wettable (table 2).

It is not difficult to render nylon unwettable by treatment with a silicone solution. We have elected to use an ether solution of silicone which renders only a temporary effect, rather than the standard textile method of producing long term water repellancy by the use of an acid catalyst. It has seemed to us that the

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TABLE 1

*Comparative tensile strengths (in grams per denier)**

Nylon *7.8*
Dacron *4.5*
Orlon *3–4.5*
Vinyon N *3*
Teflon *1.25–1.5*

* From Textile Handbook—Milton Harris. Washington, D. C.

TABLE 2

Water absorption chart (Immersion at 25C.)*

Nylon 4%
Dacron *0.1% or less*
Orlon *1%*
Vinyon N *0.1% or less*
Teflon *0*

* From Textile Handbook—Milton Harris. Washington, D. C.

characteristic of water repellancy is only important for 2 or 3 minutes until the pores in the fabric have become sealed with thrombus and that late water absorption would have little effect.

With this background, a braided nylon tube has been chemically modified in our effort to meet certain of these characteristics of an "*ideal graft*". As previously described[1] braided tubes have been dipped in formic acid solutions of various concentrations, causing each thread to swell and adhere to adjacent threads. This reduces the pore size, reduces the tendency to fraying of the cut ends of the tube, and produces a tube that is relatively noncollapsible in its cylindrical shape. By pushing the ends of the tube together on a glass rod a permanent circular crimp is produced that allows the tube to flex as much as 180 degrees without kinking. We were intially quite disturbed by the internal irregularities produced by these crimps, since all previous indications had led to the assumption that vessel linings must be quite smooth to prevent turbulence and thrombosis. Nevertheless, a large number of small caliber (6 mm.) tubes were tested in the abdominal aorta of dogs and a very low incidence of thrombosis resulted.[1] It was found that distention of the tube by arterial pressure partially obliterated this irregularity and that early lining thrombosis along the inside of the tube filled in the remaining uneven surfaces in a very few minutes after blood flow was restored. Before use, all formic acid is carefully removed and the tube is treated with a silicone dip.

Early clinical experiences with stiff straight nylon tubes, even in areas where no flexibility was necessary were discouraging.[2] These rigid tubes would not easily conform to body contours and the thrombosis rate was high. Therefore, only crimped tubes, made more flexible by using more dilute formic acid solutions, are now used. It is necessary to heat-seal the cut end of these softer tubes with a cautery to allow the secure purchase of an arterial stitch. This can easily be accomplished with an electric pistol-cautery at the operating table.

Twenty such crimped grafts have been inserted as arterial replacements or

by-passes in the iliac, femoral, popliteal and carotid arteries. The first 10 cases have been reported in detail.[2] Several have been in place over 1 year and are still patent, but the long term ability of this type of prosthesis to remain open is yet to be determined, especially in long small caliber vessels.

The following 2 cases, previously unreported, are described to illustrate certain possibilities and pitfalls in the use of this type of graft.

CASE REPORTS

Case 1. E.B. Aged 64. This man had suffered from calf claudication for 2 years prior to admission. One year before entry he had a bilateral lumbar sympathectomy at another hospital. This did not relieve his claudication; in fact it became somewhat worse and pain appeared at a higher level following this procedure. When first seen he was complaining of constant severe pain, most severe in the region of the buttocks extending down the posterior thighs and ischemic burning pain in both feet. He was requiring demerol every 3 hours by the clock for relief of this pain and had been using such medication for about 3 months. He could walk very slowly, but any walking at a normal pace would increase the severity of the pain in his buttocks and down the legs. On physical examination this tall, thin man was in obvious pain and very emaciated. There were no palpable pulses in the lower extremity at all. A strong abdominal pulsation could be felt just below the xiphoid

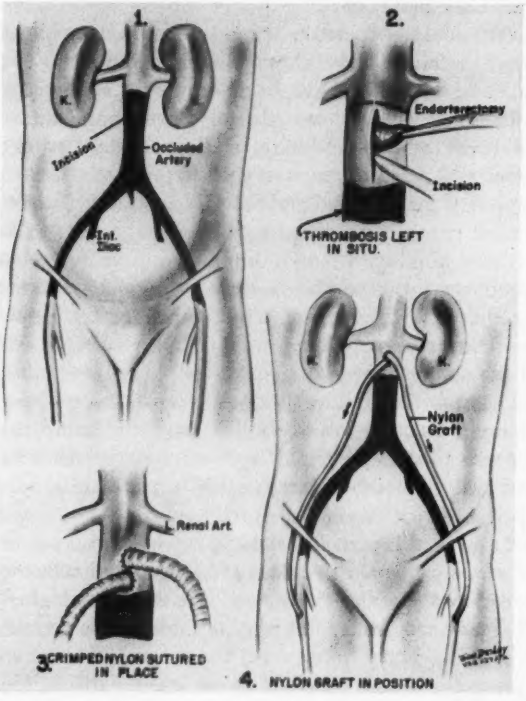

FIG. 1 Case 1. Diagrammatic representation of the patient's aortagram and of the operative procedure of aortic-femoral by-pass described in the text.

but none below this level. The legs were both atrophic and appeared quite reddish purple on dependency, this color change extending up to the midcalf. The skin of the feet was glossy and thin and there was no hair growth from the knee down. Both feet were extremely cold. It was thought that this patient had diffuse obliterative arteriosclerosis and that the likelihood of segmental obstruction was quite small. Nevertheless, at the insistence of the patient and his physician, a lumbar aortagram was carried out. The obstruction began as anticipated at the level of the renal arteries and there was complete obstruction of the entire aortic bifurcation including the common and external iliac vessels as well as the hypogastric on each side. Numerous small collateral channels were visualized in the pelvis, none of any great size. Much to our surprise, a second film, taken 2 seconds after the first exposure to include the vessels of the thighs, revealed normal caliber superficial femoral vessels extending all the way down to the knees (fig. 1). On the basis of this aortagram finding the patient was explored, first through two incisions in each groin over the superficial femoral arteries. These were found to be sufficiently soft and pliable to warrant the continuation of the operation. A left paramedian incision was carried out and the aorta was exposed just below the renal arteries. It was freed up for a distance of 2 inches below the renal branches and a longitudinal incision made in the anterior wall of the aorta about 2 cm in length. There was no pulse in the aorta at this point and calcific plaques could be felt through the wall. A thromboendarterectomy was completed, a clamp was applied just below the renal vessels and two long crimped nylon grafts, each with an internal diameter of 9.4 mm. were sutured one above the other into the longitudinal incision of the aorta. These grafts were then brought down the pelvic wall without burying them beneath peritoneum and brought through a tunnel in the femoral canal out to the groin incision where they were each sutured end to side to the superficial femoral vessels. After the release of all the clamps there was a moderate amount of bleeding through the pores of the two grafts amounting to about 200 cc. blood loss before this ceased. Good blood flow was obtained in the femoral artery below the graft on the left. There was only a small pulse immediately in the femoral vessel on the right and this slowly diminished in intensity and finally disappeared. Clots could be felt developing in the right aortic-femoral graft. Incisions were made to open up the lower anastamosis. This was unsuccessful as the graft reclotted on several occasions as it was being watched. Finally it was decided to remove this graft completely after dissecting further down the right superficial femoral and finding a softer and more pliable area about 3 inches below the profunda branch. A second crimped nylon graft of the same caliber was sutured to a longitudional incision in the superficial femoral artery on this side, brought through the femoral canal and sutured to the cut end of the previous graft at a point about 1 inch from its junction with the aorta. This graft to graft anastomosis was easily carried out after heat sealing the two cut ends and the thrombus in the previous graft was carefully aspirated before allowing blood flow to resume. Clamps again were removed and a strong pulse then was felt below the junction of the graft with the femoral artery. All incisions were closed and the following day strong pedal pulses were felt in both feet. No postoperative complications developed and the patient was able to walk without difficulty in about 7 days. One of the most interesting aspects of this particular case was the fact that the constant severe rest pain in the region of the buttocks completely disappeared; also he no longer had claudication in the gluteal regions after blood flow had been re-established in the femorals. The narcotics were gradually eliminated and this patient was able to return home without sedation and has been doing well for 4 months.

Comment: This patient was instructive to us from several standpoints. The presence of constant severe ischemic rest pain and dependent rubor does not always indicate inoperable diffuse obstruction as we had previously come to believe. It was instructive from the standpoint, also, that one must dissect as far as is necessary to obtain a normal vessel distal to the obstruction if adequate run-off is to be obtained and thrombosis avoided. Every effort was made in both these distal anastamoses to carefully reapproximate the intima at the point of longitudinal incision into the femoral arteries. The cases of the

initial failure on the right side is believed to be due to attempting to insert the graft too far proximal in an area where the femoral artery was thickened and the lumen too narrow. The third interesting observation on this patient was the disappearance of pain in the buttocks on the resumption of blood supply to the femoral arteries. We had warned the patient prior to surgery that the vessels in his pelvis might be too diffusely obstructed to be helped by grafts which would increase blood supply to his legs. His improvement would seem to be evidence that retrograde flow up the femoral artery and collaterals was allowing better blood supply to the gluteal region.

Case 2. Mr. J.P., aged 53. This patient suffered from calf claudication in both legs for a period of 5 years with gradual progression until he could walk less than ½ block without stopping to rest. On examination femoral pulses were strong bilaterally but there was a loud thrill felt over the left femoral pulse and there were no popliteal or pedal pulses in either leg. The patient's symptoms were worse in the left leg; there were no trophic changes in the feet and no color changes on elevation or dependency. A lumbar aortagram was carried out because of the presence of the thrill in his left femoral, followed by a second exposure 2 seconds after the first to include the femoral vessels of the thigh. Figure 2 shows an outline of the vessels visualized in these roentgenograms. A very narrow area of stenosis was found in the external iliac on the left; a long complete obstruction of the superficial femoral in the left thigh was seen with fairly good collaterals, especially through the profunda branch of the femoral. On the right side, a shorter complete obstruction of the superficial femoral was apparent but the middle third of the femoral was quite narrow, gradually widening out into a fairly normal caliber popliteal artery. The left external iliac was explored first through a left oblique extraperitoneal incision and a thromboendarterectomy was carried out on a segment about 1 inch in length in the area of stenosis. The intima was carefully tacked back to the muscularis and adventitia at the lower end of this end-

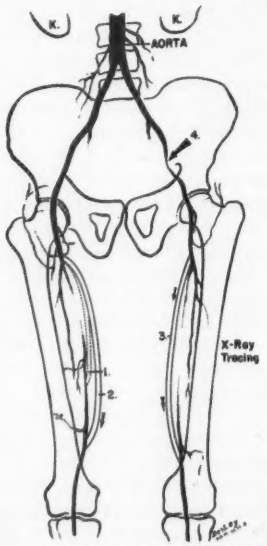

FIG. 2 Case 2. Diagrammatic representation of the arteriographic findings and operative procedures carried out on J.P.: (1) initial nylon graft in right femoral by-pass; (2) second nylon graft sutured lower to the more pliable popliteal artery; (3) left common femoral-popliteal by-pass graft; (4) area of external iliac stenosis treated by endarterectomy.

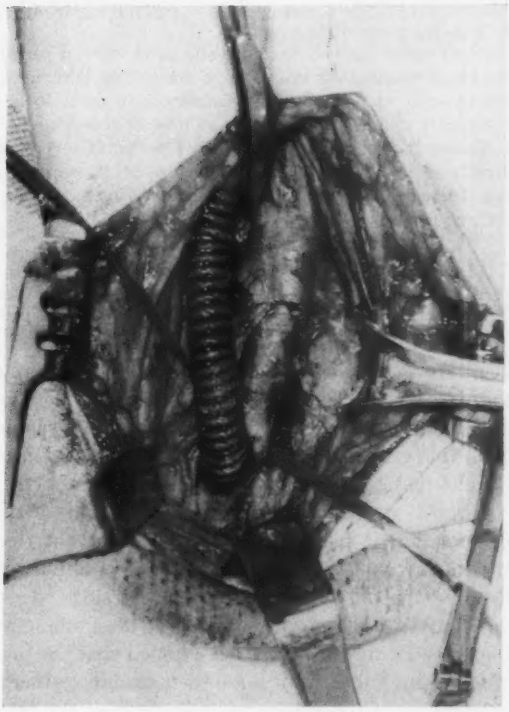

FIG. 3 End to side anastomosis of crimped nylon graft to common femoral artery

arterectomy. A long by-pass graft then was inserted into the left thigh anastomosing a crimped nylon graft with 7.7 mm. internal diameter to the common femoral artery above and to the popliteal artery below the obstruction. The patent vessels were exposed as previously described through two separate incisions and the graft was pulled subfascially between these two incisions. A similar by-pass graft was inserted into the right common femoral artery and anastomosed to the superficial femoral in the area of the midthigh. No pulse appeared below the graft at this level after its insertion but all incisions were closed and the patient returned to his bed. The following day there were very strong pulses in the left foot, none in the right. Three days later the patient was returned to the operating room and the incisions in the right leg were reopened. The graft was found to be thrombosed, as anticipated. This graft was removed except for 1 inch of the proximal end which was carefully cleaned out with a sucker and clamped temporarily at its junction with the common femoral artery. A new graft of the same diameter was pulled subfascially along the same channel as the previous one, but this time the distal end of the graft was anastomosed to the popliteal artery well down the leg and in the area where the artery was much softer and more pliable. The anastomosis was quite easy to perform at this level and the upper end of the new graft was sutured end to end to the stump of the previous graft, after heat-sealing the two cut ends. After removal of clamps the blood flow was immediately restored and a good popliteal pulse could be felt below the junction of the graft with the artery.

Pedal pulses were strong the following day and have remained strong for a period of 3 months. The patient's claudication disappeared.

Comment: This patient again showed the importance of careful judgment as to the proper location of the distal anastomosis in an area of artery that is sufficiently pliable and with sufficient lumen to carry enough run-off. Mistakes were made in both these cases, in trying to suture the graft too far proximally in an area of stenosis where run-off down the vessel was poor. A second attempt was satisfactory after going several inches down the vessel to a more patent and pliable area. This patient also points out the necessity of occasionally using thromboendarterectomy for short segments of narrowing or stenosis in conjunction with grafting procedures.

DISCUSSION

These 2 cases have presented some of the possibilities as well as some of the pitfalls in the use of any graft. It is our belief that the acute thrombosis of almost any type of artificial artery is more likely due to technical errors or severity of disease of the artery rather than to faults of the graft itself. The important consideration in regard to the type of graft chosen for arterial replacement is the length of time which this graft will stay open in respect to the number of years observed. It is thought that large caliber grafts regardless of type such as those in the aorta and iliacs will stay open for many years because of the size of their lumen. On the other hand, it has already become apparent that long, small caliber grafts in the thigh will show a greater likelihood of late thrombosis within a period of 1 or 2 years. It is not possible at this time to state whether or not the prefabricated nylon grafts will stay open any better than vein grafts or arterial homografts or collapsible fabric tubes. We have hoped that the property of noncollapsibility of these long tubes may tend to maintain patency longer than collapsible veins or arteries. Our longest by-pass graft at the present time has been in place only 6 months, so we are in no position to judge the long-term effectiveness of this particular material. It is believed that greater improvements can be made in this prefabricated type of graft by using a finer yarn which will be easier to handle and suture and still be just as noncollapsible and just as effective as a blood conduit. Efforts in this direction are being continued. Larger caliber crimped tubes are now available for making permanent by-pass around the thoracic aorta, if such should be necessary for removal of aortic aneurysms. A method is being developed to incorporate branches at right angles to the sides of such crimped tubes for the replacement of segments of aorta including renal arteries and other branches. There seems to be no end to the possibilities of prefabrication. The chief advantage at the present time of homografts is the case of suturing and there seems to be no reason why this advantage cannot eventually be built into the prefabricated tubes.

SUMMARY

Some thoughts on the characteristics of an *"ideal"* fabric graft were discussed. Efforts to produce a tube with some of these characteristics were described. Two

clinical cases of aortic-femoral and femoral-popliteal by-pass grafts were discussed in detail pointing out certain possibilities and pitfalls in the use of crimped nylon arterial grafts.

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RUPTURE OF ABDOMINAL AORTIC ANEURYSM INTO INFERIOR VENA CAVA: SUCCESSFUL SURGICAL REPAIR

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Most arteriovenous fistulas result from trauma. A smaller number occur on a congenital basis. Other causes are less frequent. It is the purpose of this report to describe a case in which a large arteriosclerotic abdominal aortic aneurysm ruptured into the inferior vena cava and in which it was possible to close the fistula, resect the aneurysm, and restore arterial continuity by a grafting procedure.

CASE REPORT

The patient was a 54 year old white man who was admitted to the hospital on Feb. 21, 1956 because of dyspnea and edema of 1 month's duration. Five years before he had discovered a mass in the upper abdomen. It caused no symptoms and, although he noted its progressive enlargement with the passage of time, he continued his work as a laborer. By January 1956 the mass seemed to occupy the greater part of the left half of the abdomen. On about January 20 there was an onset of dyspnea on slight exertion, orthopnea, and marked swelling of the lower extremities, abdomen and back. He became anorexic and gained weight in spite of poor intake of food. He was placed on digitalis and diuretics, and, even though considerable diuresis occurred, he failed to improve appreciably. After a period of hospital treatment without noticeable benefit, he was admitted to our institution. The pulse rate was 94 and the blood pressure 140/54 to 0. He weighed 140 pounds and appeared poorly nourished. The veins were distended, the abdomen protuberant, the lower extremities markedly edematous, and there was pitting edema over the sacrum. The liver edge was palpable 5 cm. below the costal margin and a fluid wave and shifting flank dullness were present. A large pulsating mass occupied most of the left upper and mid portions of the abdomen. Over it a loud continuous bruit was audible. The pulse had a collapsing character. There were moist rales at both bases. The heart was enlarged to percussion, a soft systolic murmur was heard at the apex, and the sounds were faint over the base.

Urine examination was negative except for a trace of albumen. The hemoglobin was 9.9 grams per 100 cc. and the erythrocyte count 3.31 million per cu. mm. on admission; the hemoglobin was 13.4 grams and the red blood count 4.37 million a dew days later. Nonprotein nitrogen was 48 mg. per cent on one occasion, 41 mg. per cent on another. Electrolyte studies revealed the chlorides to be 92, sodium 134, potassium 6.1 and the CO_2 combining power 27 MEQ. Prothrombin time was 49 per cent of normal on admission and 74 per cent later on. Total proteins were 6 grams per cent, the albumen being 3.7, the globulin 2.3. Electrocardiographic tracings revealed nonspecific ST-T changes. Roentgenogram showed the heart to be enlarged (fig. 1, A). There was fluid at the base of the right pleural cavity and evidence of interstitial fibrosis throughout both lung fields. On abdominal films a large abdominal aneurysm could be outlined by calcification in its wall (fig. 2). Intravenous pyelograms showed prompt opacification of both kidneys but poor concentration.

Venous catheterization studies revealed the oxygen saturation in the femoral vein to be 54 per cent, that in the external iliac 50 and in the common iliac 60 per cent. The saturation increased sharply in the inferior vena cava below the level of the renal vessels to a level of

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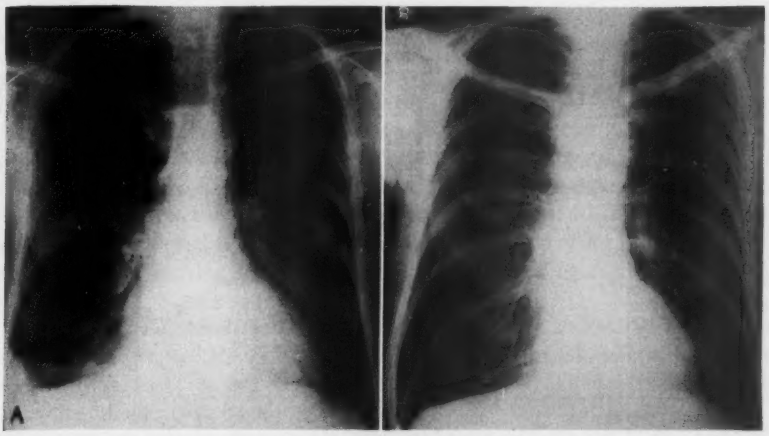

FIG. 1. Photographs of chest roentgenograms A before and B 5 weeks after operation. The postoperative film shows considerable reduction in heart size and clearing of the pleural effusion.

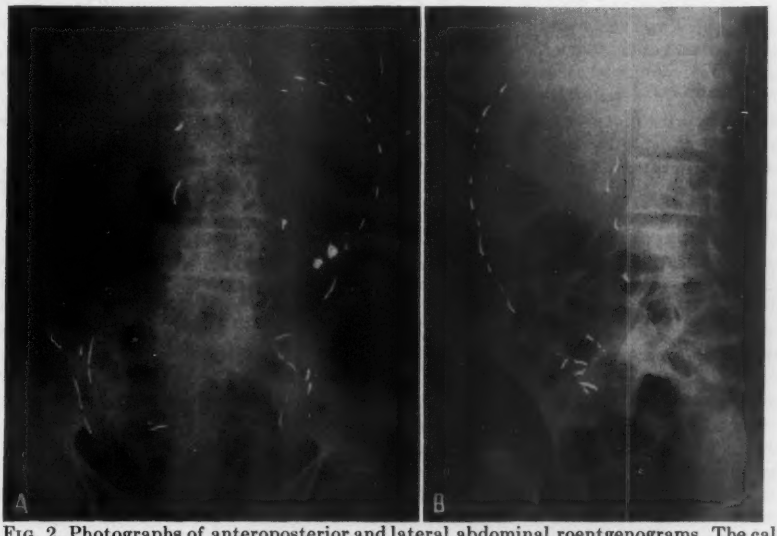

FIG. 2. Photographs of anteroposterior and lateral abdominal roentgenograms. The calcific deficits in the aneurysm have been retouched for clarity.

87 per cent. It was 85 per cent in the thoracic vena cava and 81 per cent in the right atrium. The pressures were elevated. The pressure in the right atrium was 24.5/−3.8, in the superior vena cava 11, in the midabdominal vena cava 12.2, in the iliac vein 22.2/6.2 and in the femoral vein 17/6.2 mm. of mercury. The predicted and actual values of pulmonary function studies are listed in table 1. The total lung capacity was considerably increased, the expiratory air volume decreased, the residual volume markedly increased, and the quotient

TABLE 1

Pulmonary function studies

Test	Patient's Value	Predicted Value or Normal Range	Per Cent of Predicted
Total Lung Capacity	7154	5500	130
Inspiratory Reserve Volume	2959	2850	104
Expiratory Reserve Volume	781	950	82
Residual Volume	3414	1925	177
Residual Volume/Total Lung Capacity	48%	Less than 35%	
Vital Capacity	3740	3800	98%
Alveolar Nitrogen	1.89%	Less than 2.5%	
Oxygen Capacity	15.04 vol. %	20.1—21.7 vol. %	
Oxygen Content (Vol. %)	13.97 vol. %	19.6–21.2 vol. %	
Oxygen Content (% Saturation)	91.4%	96–99%	

of residual volume and total lung capacity increased. Oxygen capacity and content were lower than normal. The total blood volume by the radio-active albumen technic was 6,600.

Only slight improvement was noted with bed rest, low sodium diet, digitoxin and diuretics. The patient's weight decreased only 4 pounds and he remained orthopneic and edematous. He was operated upon on March 6. A very large aneurysm was found to arise about 1.5 cm. below the renal arteries and to extend down to the bifurcation of both common iliac arteries. It was about 25 cm. long and about 12 or 13 cm. in diameter. The vena cava was markedly enlarged. The fistula was located about 4 cm. below the origin of the aneurysm. One could palpate here an opening between the aneurysm and the vena cava about 1.5 cm. in diameter and compression at this point obliterated the strong continuous thrill which was present. The aorta above and the external iliac and hypogastric arteries below were isolated, the left side of the aneurysm freed as much as was possible, the parent arteries were cross-clamped, and resection of the aneurysm was begun from above. It was gently freed from the vena cava down to the point of the fistula. The involved segment of vena cava was now tangentially occluded with a curved Pott's ductus clamp so as to close the fistula and leave the main vena caval pathway open. The aneurysm was severed from the vena cava and the opening in the vena cava closed by lateral suture. After the aneurysm was removed a Y shaped nylon filter graft was sutured to the aorta above and to the distal end of both common iliac arteries below with everting mattress sutures of no. 0000 arterial silk. An appendectomy was carried out. The patient stood the procedure well. His blood pressure did not drop below 120 systolic and the pulse rate did not rise above 90. Two thousand three hundred cc. of whole blood were administered during the procedure.

Convalescence was uneventful. By the following morning the edema and all other evidences of congestive failure, save for a palpable but receding liver edge, had disappeared. The patient was made ambulatory on the fifth day and discharged from the hospital on the sixteenth postoperative day. Roentgenograms made on the fourteenth postoperative day showed the heart size to be 3 cm. smaller in transverse diameter, than on the preoperative film. When seen 5 weeks after operation his weight was only 109 pounds although he was in a much improved nutritional state. This represented a loss of 27 pounds in weight from the preoperative value of 136. On this occasion the hemoglobin was 12.9 Gm., urine examination negative, NPN 40 mg. per cent, prothrombin time 85 per cent, chlorides 97, sodium 137, potassium 4.7, and CO_2 combining power 29.5 MEQ. The total blood volume, which had been 6,600 before operation and 4,900 prior to discharge, was 5,300 cc. at this time. The heart size was relatively normal (fig. 1-B.). He has remained well.

DISCUSSION

Perforation of abdominal aortic aneurysms into the vena cava have been described from time to time. In Matas' excellent review of aneurysms in Keen's Surgery, he pointed out that Bonet in 1907 had collected 20 cases in which arteriosclerotic aneurysms had perforated into the inferior vena cava. Only recently, however, have such disastrous complications been treated successfully by surgical methods. Eiseman and Hughes have reported a similar case to ours in which the fistula was closed at the initial operation and 5 weeks later the aneurysm was resected. The patient was entirely relieved of his heart failure but died some months later from a duodenal fistula resulting from his operative treatment.

In discussing Eiseman's remarks concerning this case at the 1955 meeting of the American Surgical Association, Cooley stated that he and his colleagues had successfully removed an aortic abdominal aneurysm with fistulous communication to the inferior vena cava.

SUMMARY

A case of rupture of a large arteriosclerotic abdominal aortic aneurysm into the inferior vena cava with prompt onset of classical signs of arteriovenous fistula and profound congestive heart failure is reported. Excision of the aneurysm with closure of the rent in the vena cava and interpolation of a graft were successfully accomplished.

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ACUTE APPENDICITIS IN THE ELDERLY MALE

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A. J. Ochsner, of Chicago, some 40 years ago was asked for his definition of the perfect surgeon. His reply has been said to have been, "He is that man who can diagnose appendicitis 100 per cent of the time correctly."

Appendicitis claims the lives of 7,700 people in the United States annually. Boyce[1, 2] has stated, "Appendicitis is unfinished business", despite impressive articles dealing with factors contributing to the decrease in mortality from acute appendicitis. This is true particularly of appendicitis in the elderly patient. At present 15,000,000 people in the country are over 60 years of age. This age bracket is rapidly expanding.

While the incidence of acute appendicitis is low among elderly people, the mortality rate from this disease reaches its peak in this group. Since we see more elderly patients today we should consider acute appendicitis more seriously as a cause of abdominal pain.

Forty-five cases of acute appendicitis in elderly male patients, 60 years of age and over, were reviewed at Hines Veterans Administration Hospital from 1945 to 1955. We were impressed by the lengthy duration of symptoms prior to surgery, difficulty in diagnosis, the errors of diagnosis, the high mortality rate, the high incidence of postoperative complications, and the unusual surgical pathology encountered.

Duration of Symptoms: The most favorable time to treat acute appendicitis is in the first 24 hours. In our series we found that the time interval between the onset of symptoms and the onset of treatment was considerably longer than the optimal 24 hour period. This was attributed to the patient's failure to seek earlier medical attention and to a lesser extent, failure in diagnosis (table I).

These patients were rarely aware of the gravity of their symptoms. Most of them passed off their symptoms as a mild gastrointestinal upset, and 20 per cent of them took cathartics. The elderly patient is apparently less sensitive to pain and conceals his disease until it is far advanced. They seek relief with remedies of their own. Carp and Arminio[3] stated they are subject to memory defects, mental rigidity, and mental distortion of their problem.

Errors in Diagnosis: The atypical nature of this disease in the elderly patient is often a stumbling block for the physician. Nineteen cases, or 43 per cent, were operated upon with a diagnosis other than acute appendicitis. We present this

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TABLE I

Time lapse in seeking treatment

Time	No. Patients	Per Cent
First 24 hours	6	13
25–71 hours	12	27
72 hours–6 days	18	40
7 days or more	9	20

to illustrate the difficulty encountered in diagnosing the disease which is the most common cause of the acute surgical condition within the abdomen.

Three patients developed diffuse peritonitis while in the hospital undergoing treatment for an entirely unrelated disorder. Three patients were treated with penicillin, presumably without diagnosis, by physicians prior to admission to Hines Veterans Administration Hospital. One patient was treated with analgesics for abdominal pain, prior to admission, presumably without diagnosis.

One patient presented himself to our admitting service and was rejected as not having a disease requiring hospitalization. He returned one week later with an appendiceal abscess. Another patient, not included in this series, presented a classical picture of acute appendicitis. He was operated upon and found to have a normal appendix. In the immediate postoperative period a diagnosis of multiple myeloma was made.

One man was diagnosed as having incarcerated inguinal hernia and at surgery was found to have an acute appendicitis and a sliding, indirect, inguinal hernia. One had a concomitant acute epididymitis which obscured the physical findings and delayed diagnosis for 24 hours.

Two were diagnosed as having intestinal obstruction, 2 perforated viscus, 1 acute pancreatitis, 1 acute diverticulitis of the sigmoid, 1 carcinoma of the cecum, 1 generalized peritonitis, cause undetermined. The physician is fooled by the infrequency of the condition, the unreliable atypical history, and the unusual physical findings.

Symptoms: The apparent pain in most patients was bizarre. Initially it was not well localized in 75 per cent, or 34 cases. Fifteen per cent, or 7 patients, complained of pain in the right lower quadrant in the beginning, however in only 49 per cent, or 22 patients, did the pain eventually localize to this region. It was common for this to occur only after 2 or 3 days had passed. In 18 of the patients the pain remained diffuse. It was cramping in nature in only 10 patients. Initially the pain was located in the upper abdomen in 3 patients, in the lower abdomen in 10, in the right lower quadrant in 7, and was diffuse in 17. There were isolated cases of pain in the right inguinal region, the right hip, and the right scrotum. This would indicate that the initial site of pain was not a good indication of the nature of the pathologic condition (table II).

Anorexia so commonly seen in typical appendicitis occurred in 32 patients, or 71 per cent. Nausea was present in only 22 patients, or 49 per cent. The same was true of vomiting which occurred in 22 patients, or 49 per cent. The majority

TABLE II

Frequency of symptoms

	No. Patients	Per Cent
Abdominal pain	45	100
Anorexia	32	71
Nausea	22	49
Distention	22	49
Vomiting	22	49
Constipation	12	27
Diarrhea	10	22
Chills	2	4
Dysuria	2	4

TABLE III

Findings at examination

	No. Patients	Per Cent
Tenderness of abdomen	43	96
Fever	39	87
Fever of 100 F. or more	31	69
Rebound tenderness	31	69
Localized finding right lower quadrant	29	64
Pulse rate over 100	25	56
No change in bowel habits	22	49
Muscle guarding	18	40
Rectal tenderness, mainly right	12	27
Abdominal distention	11	24
Mass noted right lower quadrant	10	22

vomited once or twice and frequently this did not occur until the second or third day. Whenever the patient complained of cramping abdominal pain there was an associated history of emesis, usually more severe and repetitious. This was true in 10 of the patients. It would appear that this was a reflex mechanism due to smooth muscle viscus, colic, or acute obstructive appendicitis.

One patient complained of alternate constipation and diarrhea. Twenty-two patients, or 49 per cent had no change of bowel habitus. Distention was a common complaint and occurred in 20 patient, or 44 per cent.

Signs: The majority of patients had findings in the lower abdomen which in most cases were pronounced in the right lower quadrant. Rectal tenderness, mainly the right rectal wall, was found in 12 patients (table III).

Concurrent Disease: Twenty-four patients, or 52 per cent, had concurrent disease (table IV) of sufficient import to warrant clinical evaluation and therapy in most cases. Some patients had more than one disease entity.

Treatment: Thirty-seven of the patients were treated surgically with a mortality in 6 of these. Eight were treated conservatively and one died. Four of the latter group were operated upon later at a more favorable time. The over-all mortality figure in this series was 16 per cent, or 7 patients.

TABLE IV

Concurrent disease, 24 cases 63 per cent

Disease	Number
Cardiovascular	
Hypertension	12
Cardiac and peripheral vascular	7
Pulmonary	
Bronchitis	2
Pneumonitis	2
Emphysema	1
Tuberculosis	1
Gastrointestinal	
Past history of peptic ulcer	3
Laennec's cirrhosis	2
Chronic pancreatitis	1
Appendiceal abscess	1
Inguinal hernia	3
Benign prostatic hypertrophy	4
Chronic nonspecific dermatitis	1
Varicose veins with ulcers	1
Syphilis	1
Anxiety neurosis	1
Acute epididymitis	1
Toxic delirium, secondary to peritonitis	1
Diabetes mellitus	2
Acute rheumatoid arthritis	1
Extreme obesity	3
Morphine addict	1

The cause of death was established at postmortem examination in all but one patient.

CASE REPORTS

Mortality Analysis:—Case 1: This was a 72 year old white man who had an acute appendicitis with a localized peritonitis. He was sick for 30 hours, on the dermatology service, where he was being treated for a nonspecific dermatitis. He was operated upon through a McBurney incision under spinal anesthesia. On the second postoperative day he developed jaundice and an ileus. He died 1 week postoperatively and at an autopsy he had evidence of an acute septicemia with a septic liver and spleen.

Case 2: A 60 year old white man was sick for 14 days with a mass in the right lower quadrant. He carried a diagnosis of angina pectoris and benign prostatic hypertrophy. He was a morphine addict and had a blood pressure of 170/100. He was treated conservatively and died on the second hospital day of an acute myocardial infarction established at autopsy.

Case 3: A 61 year old white man was sick for 48 hours and took a laxative for diffuse abdominal pain. His findings were localized to the right lower quadrant and he was operated upon through a McBurney incision under spinal anesthesia. He was lethargic postoperatively, but not alarmingly so, and died suddenly on the second postoperative day. He was thought to have suffered a cerebrovascular accident, although autopsy was not allowed.

Case 4: A 61 year old white man, sick for 48 hours, was operated upon through a right paramedian incision under general anesthesia. At surgery the exposure was difficult and he was found to have a retrocecal localized appendicitis under the right lobe of the liver.

Postoperatively he developed a pelvic abscess which ruptured on the eleventh postoperative day. He died of a generalized peritonitis.

Case 5: A 65 year old white man was sick for 48 hours while a patient in the hospital on the Medical Service. He was on cortisone therapy for acute rheumatoid arthritis with deformity. A tracheostomy had been performed on this patient for acute laryngeal edema attributed to previous cortisone withdrawal. He also had a duodenal ulcer and chronic bronchitis. He was operated upon through a McBurney incision under spinal anesthesia. A simple acute appendicitis was found. Postoperatively he developed ileus, urinary retention, bronchopneumonia and atelectasis. A suprapubic cystostomy was performed for the urinary retention. At autopsy death was attributed to generalized peritonitis.

Case 6: This 60 year old white man was ill for 24 hours. He was obese, weighing 218 pounds. Surgery was performed through a right transverse incision under spinal anesthesia. The patient had a diffuse peritonitis secondary to a perforated appendicitis. The appendix was located retrocecal and under the right lobe of the liver. Postoperatively he developed ileus and a bronchopneumonia. At postmortem examination death was attributed to generalized peritonitis.

Case 7: A 72 year old white man was sick for 72 hours. He was moribund at the time of admission and died 12 hours later. He was thought to have a mesenteric vascular problem, but at autopsy was found to have had a perforated appendicitis with generalized peritonitis.

Pathology: Only 11 patients, or 24 per cent, had simple acute appendicitis without adjacent peritoneal contamination. Sixteen patients, or 36 per cent, had a localized peritonitis. Of these 13 had an abscess formation. Eighteen patients, or 40 per cent, had generalized peritonitis (table V).

Those who developed generalized peritonitis usually were seen in the 48–72 hour period. Those who walled-off, or remained localized were generally seen at a later date. These were patients who came in 1 week later, or even longer in some cases.[10]

The clinical picture very often does not give a true concept of the actual pathologic condition. In obstructive appendicitis the part distal to the obstruction distends with gas and pus. The vessels, frequently sclerotic, in elderly patients, occlude rapidly. Necrosis occurs more rapidly as a result and bacteria penetrate through to the peritoneum with or without visible perforation. There is a sudden rupture of the distended or gangrenous appendix with a spread of infection into the peritoneal cavity. (This process is worse in the elderly patient because the peritoneum is frequently unprepared. There is a much poorer response in the geriatric patient.)[7,9]

In the younger patient a more gradual spread of the infection promotes a fibrinoplastic reaction which localizes it and shuts it off from the general peritoneal cavity. When this occurs in the older patient, abscess formation is frequent and because of a diminished response to pain he seeks medical care at a much later date.

Carp[3] attributes a poor response to a lack of lymphoid follicles in the appendiceal wall resulting in a limited local resistance. Arteriosclerosis of the blood vessels results in ischemia and a rapid gangrene. Fecaliths, foreign bodies, and fibrous tissue, strictures from previous infection and a diminution in normal blood volume add to the abnormal state.

TABLE V

Pathology

	No. Patients	Per Cent
Appendicitis	45	100
Simple acute	11	24
with localized peritonitis	3	7
with abscess formation	13	29
with generalized peritonitis	18	40

TABLE VI

Postoperative complications, 22 cases 59 per cent

Complication	No. Patients	Per Cent
Ileus	16	43
Wound infection	6	16
Wound hematoma	3	8
Urinary retention	2	5
Cardiac failure	1	3
Postoperative hernia	1	3
Rupture pelvic abscess	1	3
Pleural effusion	1	3
Cerebrovascular accident	1	3
Foreign body granuloma	1	3
Jaundice	1	3
Difficult diabetes, management	1	3

Aging itself changes the reaction of the individual to disease. There is a decrease of sustained antibody production and tissue defense mechanisms are lowered. There is impairment of tissue nutrition and circulation. The elderly patient has less homeostatic efficiency and lowered nutritional reserve. His metabolic processes are slowed and the response of body distress is impaired.[4,5]

Postoperative Complications: Many of the patients had more than one complication. The choice of anesthesia, choice of incision, and the use of drains did not seem to play an important part in the eventual outcome of cases in our series (table VI).

CONCLUSIONS

Fifteen million people in the United States are over 60 years old. As this age bracket continues to expand, the problem of appendicitis in the elderly patient will increase accordingly. Early diagnosis and early treatment is necessary to reduce complications and mortality. Delay by the patient in presenting himself, delay in diagnosis because of atypical history and physical findings, underlying degenerative disorders, high incidence of far advanced surgical pathology with relative lack of symptoms make for a long interval between the onset of the trouble and treatment. For acute appendicitis is relatively infrequent in the elderly patient, but the physician must be mindful of the atypical clinical picture[6,8] and bear in mind that appendicitis is still unfinished business.

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DISCUSSION

I wish to compliment Drs. Gillesby and O'Reilly on such a careful analysis and a very interesting presentation. Certainly appendicitis is, and will remain, "*unfinished business*". We will be dealing more and more with this disorder in aged people in the future. It is quite timely that we again be reminded of the peculiarities of this disease in the aged.

At the University of Arkansas Hospital, we have seen 20 cases of acute appendicitis in the last 5 years in patients over 60 years of age. There were 2 deaths for a mortality of 10 per cent. Seven patients were over 70, the oldest being 78. Nine were Negro and 11 were white. There were 14 males and 6 females, which indicates acute appendicitis remains primarily a disease of males even in the older age group. In general, our experience closely parallels those of the authors. Symptoms were frequently bizarre, diagnosis difficult, and complications common. Only 1 patient was seen during the first 24 hours of illness. Appendectomy was performed on 12 patients from the second to fifth day after onset. All except 4 had localized or generalized peritonitis, indicating the virulence of this disease in this age group. A right lower quadrant abscess was drained in one patient 15 days after onset of illness and 24 hours after admission to the hospital. The 2 deaths in the series occurred in this operative group, one patient succumbing from peritonitis and another from a cerebral vascular accident. Seven patients presented themselves from 5 to 14 days after onset of illness with a right lower quadrant mass and subsiding symptoms. These patients were successfully treated on a nonoperative regime. Four have subsequently had interval appendectomies.

The possibility of acute appendicitis must be seriously considered in every elderly patient with abdominal pain. Unfortunately because of factors previously outlined, the majority of these patients will continue to seek medical advice only after several days of illness. One cannot procrastinate with nonoperative measures at this time because of the fulminating course of the disease, the lessened ability to combat peritonitis, and the increased tendency for complications in this age group. The appendix should be removed. On the other hand, special care must be exercised preoperatively to correct dehydration, electrolyte imbalances, decreased blood volume and anemia. Cardiorespiratory and renal reserve are apt to be seriously impaired. Spinal anesthesia is preferred if not contraindicated. Postoperatively, measures to prevent atelectasis, thrombosis, and overloading of the circulation or kidneys should be carefully observed. Early ambulation and ridding the tracheobronchial tree of secretions are particularly important.

Patients seen after the first 5 to 7 days with a localized right lower quadrant abscess should in general be treated nonoperatively. In most instances, the process will continue to subside spontaneously and interval appendectomy can be performed at a later date. If improvement is not prompt, the abscess should be gingerly drained.

A high degree of suspicion is necessary in this problem. The only consistent symptom of acute appendicitis in this age group is abdominal pain.

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LATE SEQUELAE OF TRAUMATIC EVENTRATION OF THE DIAPHRAGM AND THEIR SURGICAL MANAGEMENT

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True eventration of the diaphragm is a situation in which a whole leaf of this intact structure is elevated high into one hemithorax. It is a relatively rare finding in infancy and is infrequently encountered in adults. Majors Kinzer and Cook[3] reported an incidence of 35 cases of congenital idiopathic eventration of the diaphragm encountered during routine roentgenographic examinations of the chests of 412,149 inductees in World War II.

In infants the symptoms usually are those of respiratory embarrassment, dyspnea, and even cyanosis. The condition, however, can be entirely asymptomatic and present as an accidental or incidental roentgenographic finding. In the adult the symptoms may arise from the gastrointestinal, circulatory, or respiratory systems. Patients may complain of vomiting, upper abdominal pain, dyspepsia and eructation of gas. Some may complain of severe dyspnea, cough, wheezing, tachycardia, and cyanosis. Regardless of cause, the abnormality is a serious one since it can produce distressing cardio-respiratory symptoms and even cardiac failure.

The physical findings are predominantly those of dullness over the side on which the diaphragm is high, or tympany if the abdominal viscera have risen high under the thoracic cage. The heart may be displaced to the opposite side.

The diagnosis is almost invariably made by roentgenographic examination of the chest and the findings are quite typical. There usually is a conspicuous elevation of the diaphragm which is in continuity. The motion of the diaphragm usually is absent or paradoxical but in all situations the contour usually is well outlined and the contents of the abdomen are apparent beneath the involved leaf.

In contradistinction, diaphragmatic hernias, which may present as a problem in differential diagnosis, are not easily outlined and only a portion of the leaflet may be seen by either roentgenographic or fluoroscopic examination. The diaphragm usually is not elevated and does not have paradoxical motion. Herniated small bowel, stomach or colon can be demonstrated by barium examination to protrude through the ruptured diaphragm.

The exact etiology of eventration of the diaphragm is poorly understood but practically all cases reported in the past have been considered congenital in origin. In a review of the literature I could find no report of eventration of the diaphragm resulting from trauma. In light of the overwhelming evidence presented in the past indicating that all eventrations are congenital in origin, the following case reports are presented because they suggest, if not prove, that eventration can be the direct result of trauma and can produce serious symptoms which may require surgical intervention.

From the Department of Surgery, University of Cincinnati College of Medicine and the Cincinnati General Hospital.

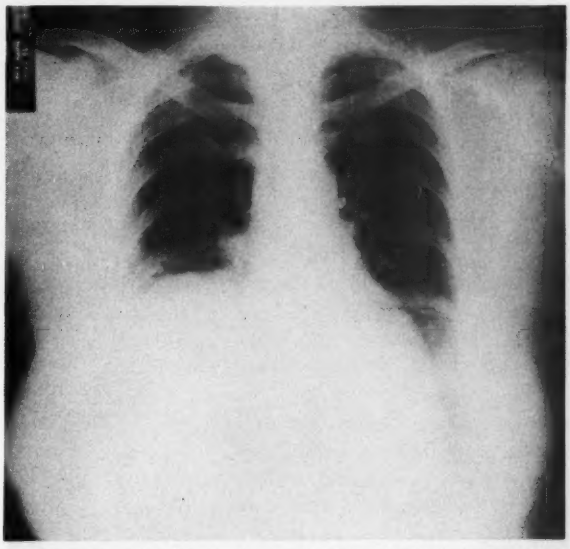

FIG. 1. (Case 1.) Paralyzed right diaphragm is at level of third rib anteriorly on right.

CASE REPORTS

Case 1: Mrs. M. S., 64 year old white woman, was first seen in consultation on Sept. 11, 1951, because of dyspnea, hacking cough, and roentgenographic findings suggesting atelectasis of the lower lobe of the right lung. The patient gave no history of chest disorder in the past and stated that her dyspnea began after a fall down a flight of stairs in April 1950. A roentgenogram of the chest at that time had reputedly demonstrated a normal position of the diaphragms. Unfortunately, the films were lost and were not available for review.

Repeat roentgenograms of the chest in September 1951, (fig. 1) showed the right diaphragm to have risen to a high level opposite the third rib on the right anteriorly and there was compression atelectasis of the lower and middle lobes of the right lung. Fluoroscopy showed paradoxical motion of the right leaf.

Bronchoscopic examination on September 13, showed normal findings except for severe compression of the right middle and lower lobe orifices which were reduced to slit-like apertures. All portions of the tracheobronchial tree, however, were definitely mobile and showed no evidence of fixation as would occur in carcinoma.

Because of the increasing severity of the dyspnea so that the patient was literally bedridden, operation was decided upon and performed October 17. The chest was entered through the bed of the eighth rib. The diaphragm which appeared to be entirely normal and had the usual aponeurotic and muscular components was at an abnormally high level near the third rib anteriorly. A right phrenic nerve was definitely present and appeared to be normal in size. There was total compression of the right middle and lower lobes of the lung as a result of the high position of the diaphragm. The right leaf was reduced down to a normal level near the eighth rib by 3 superimposed rows of *"reefing"* mattress sutures of medium silk. By this technic a cuff of diaphragm approximately 6 inches in length was turned inferiorly between the diaphragm and liver. Following this procedure positive pressure resulted in complete reexpansion of the middle and lower lobes of the lung (fig. 2).

The patient did well and was discharged home on her tenth postoperative day. Four years and three months after operation the patient remains free of dyspnea and leads an active life.

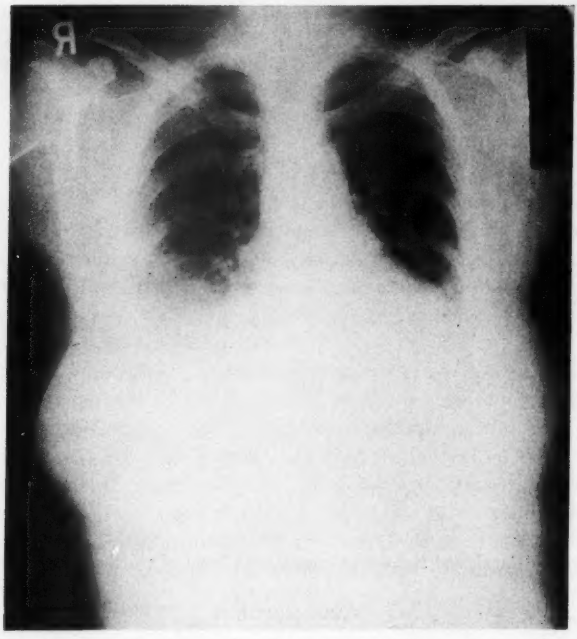

FIG. 2. (Case 1.) Right diaphragm has been "*reefed*" down to normal position. Right middle lobe and right lower lobe reexpanded.

Discussion: In view of the history of normal roentgenographic findings in the chest and the complete absence of dyspnea prior to this patient's injury and in view of the subsequent roentgenographic evidence of high position of the right diaphragm, it is a logical conclusion that the eventration could have resulted from some type of indirect injury to the phrenic nerve following the fall in 1950. Additional evidence that the lesion was not congenital in origin was the finding of a relatively normal diaphragmatic structure at the time of operation rather than the very thin, attenuated, atrophic leaf one would expect to encounter if this abnormality had been present since birth.

Compression atelectasis of 2 lobes of the lung resulted from the high rise of the diaphragm. Bronchoscopic examination was necessary to exclude the possibility of endobronchial disorder, such as stricture, neoplasm, or retained foreign body.

Transpleural "*reefing*" of the eventrated diaphragm gave permanent relief from symptoms.

Case 2. Mr. G. N., 63 year old white man, was struck in the abdomen by a large grave stone weighing an estimated 80 to 100 pounds. The accident occurred when the patient lost his footing while lifting the stone on April 4, 1954, and sustained a compression injury of the abdomen. He experienced sudden, acute, abdominal pain and was immediately admitted to a hospital. Exploratory laparotomy revealed a ruptured ileum which was repaired by simple suture closure. His course was uneventful and he was discharged from the hospital 16 days following surgery.

Physical examination at the time of admission to the hospital showed entirely normal findings over the chest with no abnormal areas of dullness. The diaphragmatic excursions were described as normal.

I was first asked to see the patient in consultation on June 3, 1955, because of a complaint

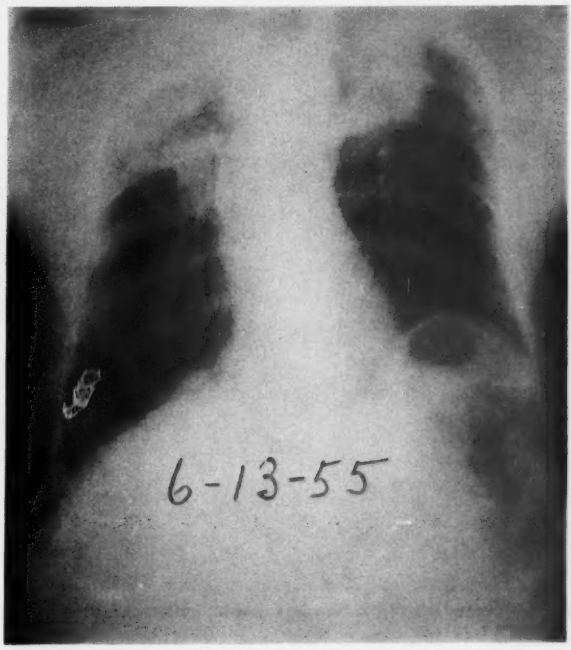

FIG. 3. (Case 2.) Traumatic eventration left diaphragm in association with benign bilateral parenchymal lesions.

of dyspnea of 6 months' duration aggravated by exertion. There was no history of cough, hemoptysis, or purulent sputum. Roentgenograms of the chest on June 13, 1955, (fig. 3) showed a high position of the left diaphragm. The trachea was shifted to the right of the midline. Circumscribed, oval shaped, solid densities were apparent over the right apex in the lateral portion of the chest and a density similar in size and shape also was apparent over the left apex near the mediastinum. The densities were thought to be the result of either pleural fibrosis or conglomerate silicosis and fibrosis resulting from the patient's occupation as a stone cutter. Carcinoma could not be excluded.

Fluoroscopic examination showed that the left diaphragm moved paradoxically. The patient presented a real problem in differential diagnosis in that he had a high left hemidiaphragm and either pleural or parenchymal lesions in both lungs which were either benign or carcinomatous.

Bronchoscopic examination on June 13, 1955, showed normal findings except for superior angulation and narrowing of the left main stem bronchus probably resulting from the high position of the diaphragm. There were superficial mucosal changes consistent with chronic bronchial infection but no evidence of neoplasm. Washings were reported negative for cancer cells and acid-fast organisms.

In view of the benign character of the parenchymal lesion and the complete absence of dyspnea until 8 months following the injury it was my impression that the high level of the left diaphragm was probably traumatic in origin. "*Reefing*" of the left hemidiaphragm was recommended if and when the patient's symptoms increased in severity.

Discussion: This patient presented a problem in differential diagnosis between carcinoma, tuberculosis, and benign lesions of the lung associated with traumatic paralysis of the left diaphragm. The injury sustained in April 1954, which was of sufficient severity to

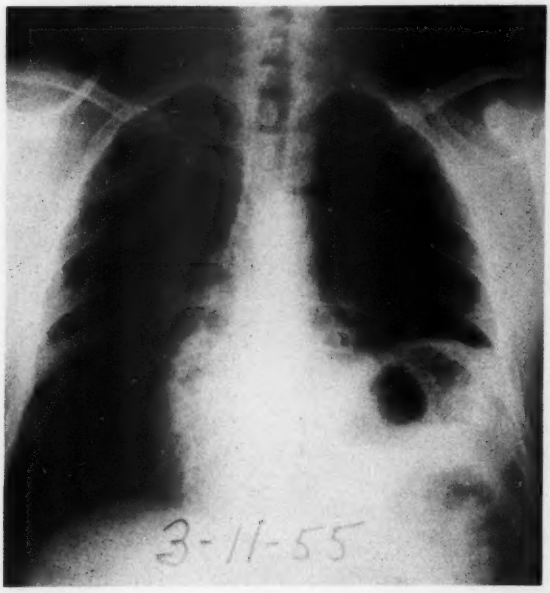

FIG. 4. (Case 3.) Traumatic eventration left diaphragm level fourth anterior rib. Note dislocation of heart to right.

result in rupture of the ileum, could very possibly have resulted in a sudden stretching of the left phrenic nerve with subsequent progressing paralysis and rise of the left diaphragm so that the patient experienced dyspnea 8 months following his injury. No roentgenograms of the chest were available prior to the abdominal injury but the physical signs at that time demonstrated normal levels and motion of the left diaphragm.

Case 3. Mr. S.A., 53 year old Negro man, was seen in consultation on Feb. 19, 1955, with a history of dyspnea which began shortly after an automobile accident in 1952 and had increased in severity up to the present time. Since October 1954, the patient had had repeated bouts of pneumonitis and more recently had demonstrated signs of cardiac decompensation. He had a constant cough productive of mucopurulent material but no blood.

Prior to the accident the patient had been completely without respiratory symptoms.

Roentgenograms of the chest on March 11, 1955, (fig. 4) showed the left hemidiaphragm at the level of the fourth anterior rib, with pneumonitis, and/or atelectasis of the lower lobe of the lung.

Bronchoscopic examination showed normal findings except for minimal compression of the basal branch bronchi of the left lower lobe. Washings were negative for acid-fast and tumor cell studies. An electrocardiogram on March 11, 1955, showed myocardial disease with left ventricular strain. The blood pressure was recorded at 190/120. A systolic murmur was audible at the apex and over the aortic area. It was the clinical impression that this patient had a traumatic paralysis of the left leaf of the diaphragm resulting in compression atelectasis and recurring episodes of pneumonitis in the lower lobe of the left lung. These disorders in turn had aggravated an already existing intrinsic cardiac disorder with intermittent cardiac decompensation. Accordingly, open chest surgery was recommended and accepted.

On March 16, 1955, the left chest was entered through the bed of the seventh rib. The left diaphragm appeared to be entirely normal except for its high position and paradoxical

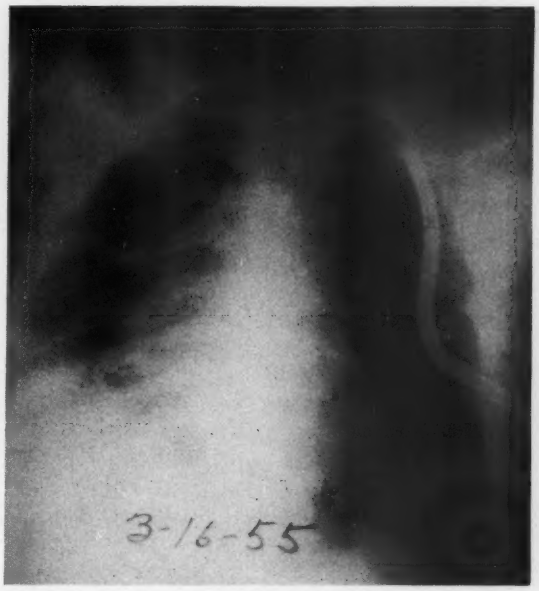

FIG. 5. (Case 3.) Postoperative roentgenogram. Note pneumothorax space left base resulting from slow expansion of lung.

motion. The left phrenic nerve presented no abnormalities except for questionable attenuation near its point of attachment to the diaphragm. The diaphragm was *"reefed"* down by 3 superimposed rows of interrupted mattress sutures of cotton so that it finally rested at the level of the tenth rib (fig. 5). Because of slow expansion of the lower lobe a pneumothorax space was present necessitating an intercostal tube and water-seal drainage. During the postoperative convalescence there was gradual expansion of the lung (fig. 6). The patient was discharged home on his twelth postoperative day, at which time his wound was healed and he was clinically improved. Subsequent follow-up studies of this patient show that he has been remarkably improved by the operation and has had no recurrences of pneumonia or cardiac decompensation.

Discussion: Eventration of the left diaphragm presented as a major complication in this patient who had known intrinsic heart disease with hypertension. It is very possible that the dislocation and rotation of the heart incident to the rise of the diaphragm and the compression atelectasis and recurring pneumonitis in the lower lobe of the left lung played a rather prominent part in producing his original episode of cardiac decompensation.

Case. 4. R.R., 53 year old white man, developed acute dyspnea in December 1954 following a compression injury of his chest and abdomen sustained when he fell from a stepladder. Previous examinations by the attending physician had revealed no abnormality in the mobility or position of the diaphragm.

A roentgenogram of the chest on Jan. 17, 1955, (fig. 7) showed the diaphragm to be high on the right and there was an associated paramediastinal density which was thought to represent a possible bronchiogenic carcinoma. Fluoroscopy of the chest showed the right diaphragm to be high and moving paradoxically.

Bronchoscopic examination revealed entirely normal findings with negative washings for acid-fast and tumor cell studies. A repeat roentgenogram of the chest on March 27 and 29, (figs. 8 and 9) approximately 9 weeks after the previous roentgenographic examination,

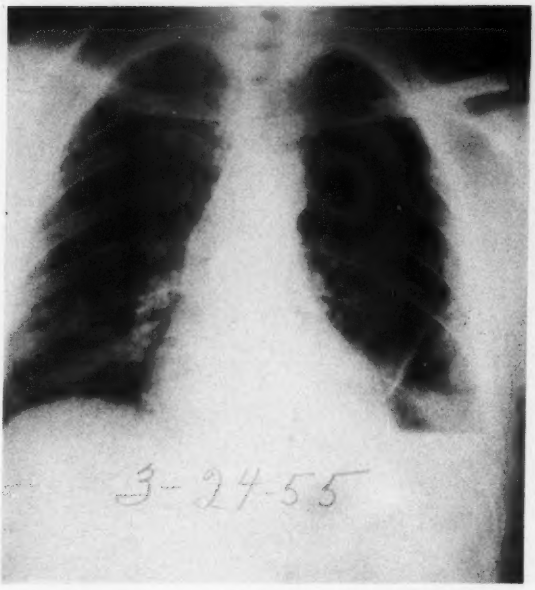

FIG. 6. (Case 3.) Roentgenogram eighth postoperative day showing conplete expansion left lung; diaphragm in normal position and heart in midline.

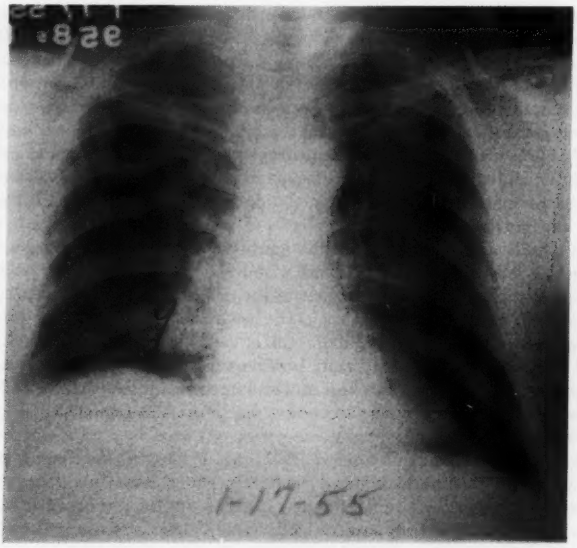

FIG. 7. (Case 4.) Posteroanterior roentgenogram showing right paramediastinal density and moderate elevation right diaphragm.

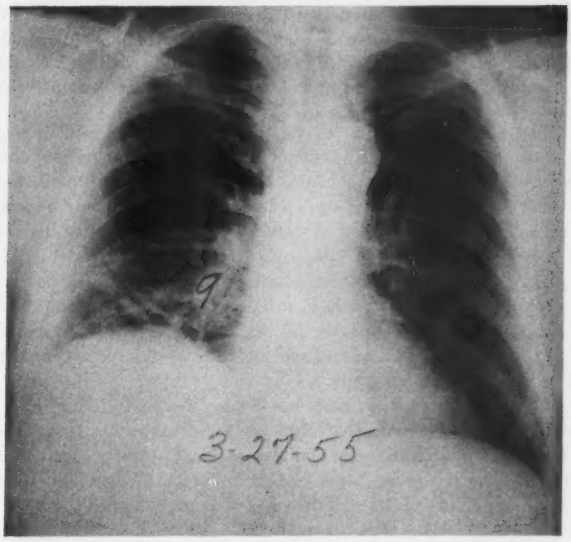

FIG. 8. (Case 4.) Roentgenogram 9 weeks later showing clearing of paramediastinal density with increasing rise of right diaphragm and atelectasis right lower lobe.

FIG. 9. (Case 4.) Note additional rise right leaf of diaphragm and disk atelectasis right lower lobe.

revealed remarkable decrease in size of the paramediastinal density on the right, but a progressive rise of the right diaphragm and compression atelectasis of the lower lobe of the right lung. Final impression was palsy of the right diaphragm with high eventration, probably traumatic in origin with hilar lymphadenitis secondary to compression atelectasis and pneumonitis.

Discussion: Traumatic eventration of the right diaphragm resulting from indirect trauma in association with chronic hilar lymphadenopathy in this patient simulated primary bronchiogenic carcinoma of the right lung with invasion of the phrenic nerve. Hospital admission was necessitated and special studies including bronchoscopy, repeat fluoroscopic and roentgenographic examinations of the chest excluded the possibility of carcinoma. Serial roentgenograms demonstrated progressive paralysis of the right diaphragm with concomitant clearing of the hilar density indicating that the patient did not have carcinoma and that the eventration was traumatic in origin. In this instance we were fortunate in obtaining roentgenographic studies of the chest during the actual development of eventration of a diaphragm following trauma. Transpleural *"reefing"* was recommended if the dyspnea persisted or increased in severity.

Case 5. Mr. C.K., 53 year old white man was in good health until July 1955, when he was struck in the abdomen by a fan blade that suddenly became detached from a factory exhaust fan. The patient sustained hematomas and lacerations of the genitals and right lower abdomen. Following the injury he developed dyspnea which increased in severity, with eructation of large quantities of gas and discomfort over the upper abdomen following his meals.

On physical examination there was very poor ventilation of the right lower lobe of the lung. The right diaphragm was high and motion was markedly decreased to absent. Roentgenograms of the chest on Nov. 25, 1955 (fig. 10) showed a high diaphragm on the right with disk atelectasis in the right lower lobe. Fluoroscopic examination showed complete paralysis of the diaphragm with paradoxical motion.

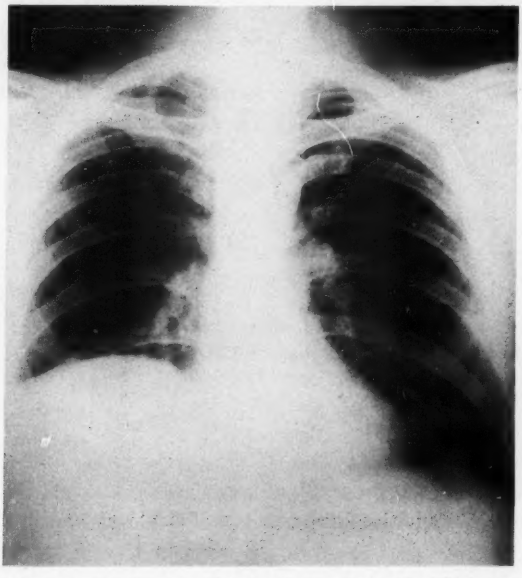

FIG. 10. (Case 5.) Paralyzed right diaphragm at level of fifth anterior rib.

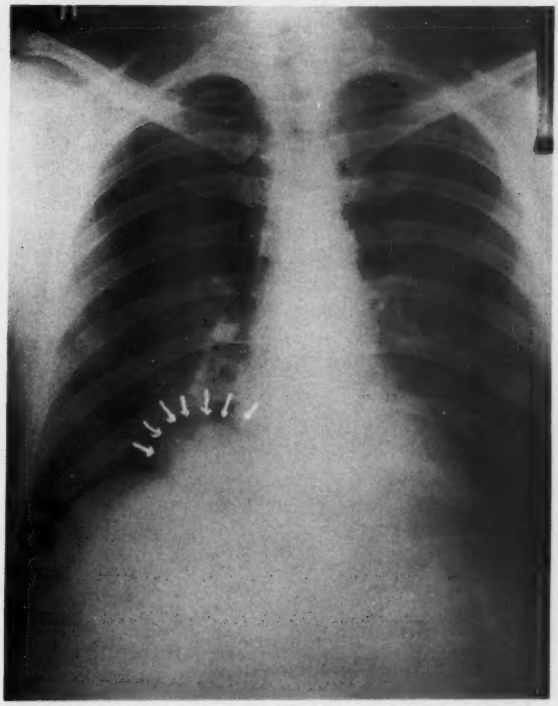

FIG. 11. (Case 6.) Posteroanterior roentgenogram chest showing herniation of liver into right chest near inferior vena cava.

A review of the patient's past clinical history in conversation with the attending physician elicited the fact that this individual had never had dyspnea in the past and that previous physical examination had disclosed no abnormality over the lower lobe of the right lung or the right diaphragm. This patient is still under observation at the present time and if his dyspnea increases or if the diaphragm continues to rise transpleural *"reefing"* will be recommended.

Case 6. R. J. W., 37 year old white man was admitted to the hospital on Jan. 10, 1954, because of the incidental finding on plain roentgenograms of the chest of an asymptomatic mass near the mediastinum above the right diaphragm (figs. 11 and 12). A diagnostic pneumothorax was unsuccessful because of obliteration of the pleural space by adhesions. Barium studies revealed no herniation of bowel. The patient had sustained a crushing injury of his chest without skeletal fractures 3 years preceding his admission. In the differential diagnosis the following lesions were considered—localized eventration of the diaphragm, primary tumor of the diaphragm and primary tumor of the lung.

Exploration of the right chest through the bed of the seventh rib was carried out on March 24, 1954. The mass was found to be a nodule of liver the size of a small orange protruding through an area of traumatic rupture of the right hemidiaphragm. The medial margin of the rupture ring extended within 1 centimeter of the inferior vena cava. Because of the size and irreducibility of the liver mass, repair of the diaphragm could not be completed until excision of the protruding liver had been accomplished. Repair of the defect then was easily completed with interrupted mattress sutures of cotton. The patient left the hospital on his ninth postoperative day.

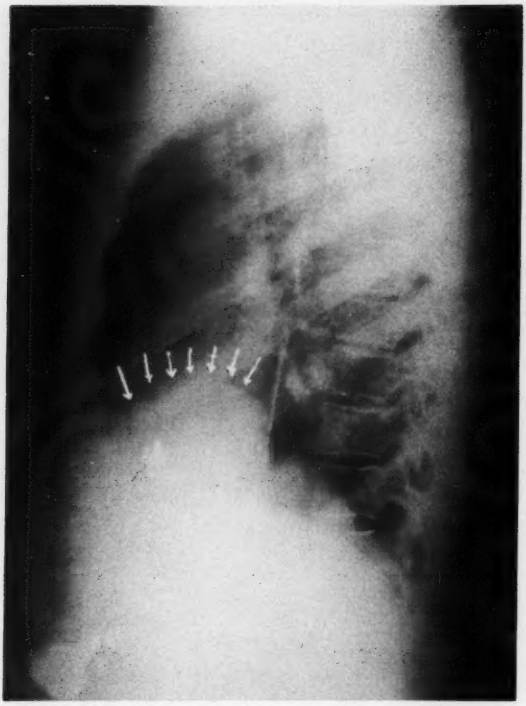

FIG. 12. (Case 6.) Lateral roentgenogram chest showing anterior position of liver hernia simulating focal eventration diaphragm.

Discussion: Rupture of the diaphragm with herniation of the liver is an unusual occurrence. The roentgenographic findings can simulate those of localized eventration of the diaphragm.

DISCUSSION

In most instances of congenital eventration of the diaphragm this structure is found to be a thin, translucent membrane containing a few muscle fibers covered by pleura and peritoneum. The phrenic nerve usually is reduced in size or may be completely absent. None of these findings were encountered in the patients operated upon whose cases are reported here. In all instances the diaphragms and phrenic nerves appeared to be normal in structure and size. In one patient there was some attenuation of the phrenic nerve near its attachment to the diaphragm. This latter finding could have been the result of traumatic attenuation as a result of acute stretching at the time of injury or actual hemorrhage into the nerve with subsequent scarring and contracture. It would be impossible to state categorically exactly what pathologic changes are responsible for paralysis of the diaphragm following indirect trauma to the abdomen or chest, but it is certainly conceivable that during a sudden convulsive tensing of the abdomen and dia-

phragm such as would occur during falls or sudden compression of the abdomen, serious stretching of the phrenic nerve with or without hemorrhage could occur. This would result in either immediate paralysis or gradual rise of the diaphragm as the areas of hemorrhage are replaced by scar tissue.

The sensitivity of the phrenic nerve to minor degrees of trauma has been well demonstrated in those instances where inadvertent pressure upon or crushing of the nerve has occurred during transthoracic procedures. The simple encircling of a normal phrenic nerve with a ligature can result in immediate total paralysis of the diaphragm. In most instances the phrenic nerve spreads out in several digitations as it inserts upon the diaphragm. A sudden convulsive contraction of the dome of this structure could conceivably result in a tearing loose of several of these strands of nerve tissue producing either local or generalized eventration of the diaphragm.

In several of the patients whose cases are reported here there was roentgenographic evidence of progressive rise in the diaphragm such as not uncommonly occurs following surgical crushing of the phrenic nerve with permanent paralysis as occurs in 25 per cent of the patients.

Special diagnostic procedures such as pneumothorax and pneumoperitoneum have been suggested to substantiate a diagnosis of eventration of the diaphragm. I believe that these procedures are not only unnecessary but are perhaps not without risk in people who already have the chief complaint of dyspnea.

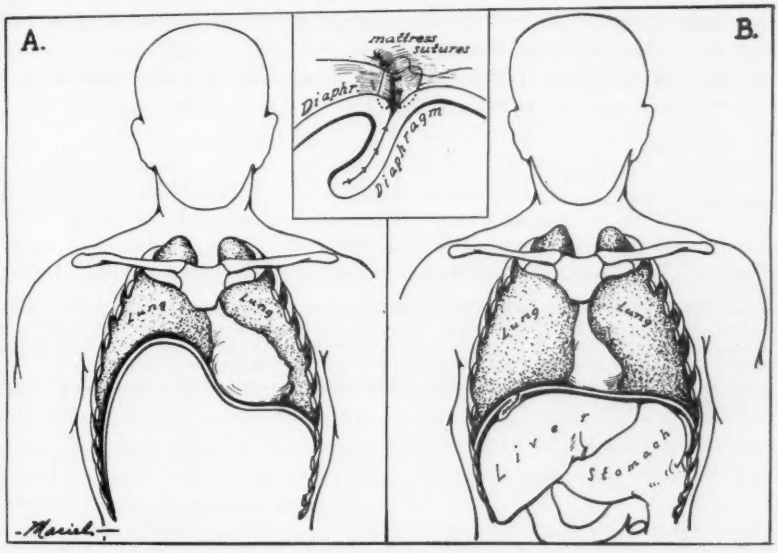

FIG. 13. *A*. Diagram illustrates compression of right lung and dislocation of heart into left chest resulting from eventration right diaphragm. *B*. Transpleural "*reefing*" as illustrated in "*insert*" permits reexpansion of compressed lung and return of mediastinal structures to midline.

Several methods for surgical correction of the eventrated diaphragm have been suggested in the past. These include transpleural "*reefing*" with superimposed rows of inverting mattress sutures[1] (fig. 13), reefing of the diaphragm after it has been pulled down into a laparotomy wound,[2] tenting and fixation of the diaphragm to the costal margin and the renal fascia, cutis grafts, repairs utilizing tantalum mesh[4] and, finally, incision of the eventrated diaphragm with either excision of the excessive portions or overlapping of the flaps by imbrication. Good results were obtained in the series of cases reported here by simple transpleural "*reefing.*" This approach permits direct inspection of the lung which in many instances is compressed or atelectatic and allows free dissection of pleural adhesions which may be numerous.

Bronchoscopic examination should be performed before any transpleural operation for eventration of the diaphragm to exclude the possibility of endobronchial occlusion.

SUMMARY AND CONCLUSIONS

Case reports have been presented which suggest, if not prove, that eventration of the diaphragm can be traumatic in origin.

Traumatic eventration of the diaphragm can simulate inoperable bronchiogenic carcinoma when the diaphragmatic paralysis is associated with otherwise benign lesions of the paramediastinum, pleura or lung parenchyma.

Serious derangements in cardiorespiratory function can result from eventration of the diaphragm even at very late intervals after the original injury and may require surgical correction of the disorder.

Transpleural "*reefing*" of the eventrated diaphragm is a safe surgical procedure that uniformly gives excellent results.

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SURGERY IN PULMONARY TUBERCULOSIS: THE PROBLEM OF THE POOR-RISK PATIENT

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The excision of significant disease residua with preservation of relatively normal pulmonary tissue represents the most satisfactory form of the surgical treatment of pulmonary tuberculosis today. Unfortunately there are many patients with persistent disease for whom excisional surgery or even the less precise and older collapse measures present prohibitive operative risks. There is some indication that this problem may be expected to increase even though one gains the impression that the volume of surgery for pulmonary tuberculosis is decreasing. This paper is based on experiences with 170 patients who were subjected to major surgical procedures in treatment of pulmonary tuberculosis between November 1953 and January 1956.

A primary factor contributing to the numbers of poor-risk patients paradoxically is the effective use of the antituberculosis drugs. It is true that drug therapy has permitted the recovery of many patients without the need of surgical help. It is equally true that many patients have improved sufficiently so that less extensive surgery is required. More significant to the purposes of this paper is the fact that effective drug therapy results in the salvage of patients who formerly would have succumbed to their disease but now reach a precarious chronic invalidism in which their disease is neither immediately dangerous to life nor safely inactive and controlled.

Another factor contributing to the difficulty of surgical treatment is the increasing incidence of pulmonary tuberculosis in older people. During the period from Dec. 1, 1954 through Nov. 30, 1955 there were 330 new admissions to the W. T. Edwards Tuberculosis Hospital. Fifty-two per cent of these were over 40 years of age, 30 per cent were over 50 years of age and 6 per cent were more than 70 years old. Chronological age in itself should not be a deterrent to needed surgery. The gradual impairment of the vital systems with the passage of time does narrow the margin of physiologic reserves in the older person. A serious problem germane to this group is a weakening of the will to live when the postoperative period becomes difficult. With many years behind and, at best, a relatively short future it becomes easy to give up the struggle for recovery. The shorter life expectancy of the aged person with tuberculosis does not reduce the need for elimination of his danger as a source of infection nor does it make his relative degree of isolation more palatable.

The problem of disease processes concurrent with tuberculosis is less common than those previously mentioned but no less important from the standpoint of individual surgical risk. Diabetes mellitus, asthma, obstructive emphysema,

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obesity, hypertension and other cardiovascular diseases add to the operative risk. Proper recognition and treatment of these associated disorders will diminish their danger to the surgical patient.

The patient with extensive bilateral disease who cannot be offered or will not accept operation when his disease cannot be controlled by medical measures presents a serious problem. The sanatorium regimen may allow his maintenance in apparent relatively good health, often with negative sputum. Yet there is ever present danger that tubercle bacilli will appear in the sputum or that further spread of the disease will occur. The patient cannot be returned safely to his family nor can he expect much, if any, further healing of his disease. With this bleak outlook it is not surprising that such patients leave the hospital against advice. Conversely one sees the patient whose temperament is such that during the long course of his disease he has been unable to endure hospitalization long enough to attain inactive status. Eventually he reaches a relatively hopeless stage of extensive bilateral involvement. It is clearly apparent that the answer to the patient with extensive chronic disease will come eventually not from some surgical procedure but from earlier case finding, more effective therapy and better solution of the socio-economic problems of the patient with tuberculosis.

The aims of the surgical treatment of the patient with pulmonary tuberculosis remain constant while the indications for employing such treatment have become uncertain at times during the development of drug therapy. We still wish to eliminate the danger of the patient's disease to himself and to others and, if possible, we wish to return him to a useful life. The uncertainty of the indications for surgery is due largely to inability to predict the future behavior of disease residua following long term drug therapy. Opinion varies from removing "less and less from more and more patients"[1] (attempting to remove all disease residua of potential hazard) to the discharge on continued long term drug therapy of patients with roentgenographic evidence of persistent but *"healed"* cavities.[2] Pathologic evidence may be cited to support each idea.[6, 7]

One indisputable indication for operation is the persistent production of viable tubercle bacilli in the sputum despite adequate drug therapy. Discussion concerning the relative indications for surgery in the patient whose sputum is negative is beyond the scope of this paper. In general we have based our indications on the hope of insuring the patients continued well being after drug treatment and sanatorium care have been completed.

The need for operative help may be clear but the wisdom of attempting a course which cannot be completed or of a procedure which may leave a crippled or dead patient is a matter to be decided with each patient individually. We would agree that absence of all mortality in the surgical treatment of tuberculosis, while indicating a skillful achievement, may also indicate too restrictive indications for operation. A more liberal application of surgery may result in some mortality but it will also result in the salvage of some patients whose chances of recovery otherwise will be slim.

It seems generally agreed that the surgical removal of significant areas of persistent atelectatic or destroyed lung and of cavitary portions of lung is the

treatment of choice. We have employed pulmonary resection whenever the condition of the patient was deemed reasonably favorable. Pulmonary tissue was conserved whenever possible by resorting to segmental resection. The amount of lung removed was based more on the operative findings of the extent and location of palpable disease than on the preoperative roentgenographic studies.

Such factors as extensive disease involvement, scarring and fibrosis about the hilar structures, extensive pleural symphysis and compensatory or obstructive emphysema in the adjacent pulmonary parenchyma contribute to increased difficulty during pulmonary resection and to increase in postoperative complications. In an attempt to salvage some patients who seemed to present rather excessive operative risk we have used various permanent collapse measures. Our estimate of relatively poor operative risk is based on clinical, roentgenologic, and the more routine laboratory examinations. Facilities have not been available for bronchospirometry nor even for determination of maximum breathing capacity. The use of such tests of pulmonary function will improve the accuracy of selection of patients for operation. Clinical estimate of the reserves of the patient's cardiorespiratory function has been made on the basis of the usual physical examination of the heart and lungs, observation of tolerance to mild exercise, and fluoroscopic examination of the chest. The review of all available chest films made during the course of the patient's illness is of great value since the extent of disease involvement gives an estimate of the amount of parenchymal scarring and destruction present. This may be only vaguely apparent in films made after long periods of treatment.

Thoracoplasty: In one group of 25 patients, most of whom presented evidence of their ability to inactivate and heal their disease as manifested by negative sputum and roentgenographic evidence of contracting, fibrotic disease, thoracoplasty has been done to permit further healing. Thoracoplasty was chosen in preference to resection because it could be done in stages when necessary, it was a procedure of shorter duration, and it avoided such possible complications a hemothorax, bronchial fistulas, and difficulties in reexpanding the lung which may follow resection.

In most of the patients who received thoracoplasty pulmonary function was poor due to the extent of the tuberculosis or less often to complicating asthma or obstructive emphysema. In some patients the general physiologic status was such that a staged procedure was much less hazardous than a major resection involving prolonged operative time. Rarely thoracoplasty has been done in the hope of assisting improvement to a point at which resection can be done.

In 6 patients with large apical cavities catheter drainage of the cavity was combined with thoracoplasty. During an anterior stage of thoracoplasty with removal of most of the first and second ribs and sometimes part of the third a catheter with inflatable bag was placed in the cavity through a stab wound and connected to a water seal drain. The thoracoplasty then was completed in stages at later dates through the usual posterior incision. Five of these patients have since been discharged with apparent inactive disease. One patient has persisting contralateral cavitary disease and remains hospitalized.

Thoracoplasty was done for 2 patients with complete empyema complicating therapeutic pneumothorax. Pleuropneumonectomy was thought to present excessive danger for these patients one of whom was a slender frail white woman of 60 years and the other a 48 year old Negro man. Recovery was complete in both. This complication of pulmonary tuberculosis is difficult to treat and results often are poor with thoracoplasty, excision with or without decortication or combinations of both collapse and excision. Fortunately this situation is less often seen since the use of pneumothorax has declined.

Six of these patients had positive sputum at the time of operation in spite of long term drug therapy. One of these is a white woman with little pulmonary reserve on whom a 3 rib, first stage thoracoplasty was done. She has never improved sufficiently to permit completion of the thoracoplasty and pneumonectomy is thought impossible. Her sputum remains positive. Right pneumonectomy was done in one patient whose sputum remained positive following thoracoplasty. Cardiac arrest occurred immediately following transfer of the patient from a prone position on the operating table to her bed. Resuscitation by massage of the heart was successful but she died several hours later apparently due to inadequacy of the remaining, partially diseased lung. One patient has had thoracoplasty in anticipation of subsequent excision of his disease provided improvement in the bilateral involvement occurs. The other 3 patients have been discharged with negative sputum. One patient, negative for a time before operation, developed a positive sputum soon afterward and has been intermittently positive since. This patient has asthma, very poor respiratory reserve and paralysis of the left recurrent laryngeal nerve apparently due to mediastinal lymph node disease.

Of the 25 patients on whom 52 stages of thoracoplasty were done, 4 remain hospitalized with disease in varying stages of activity, one left the hospital against advice and the remainder were discharged as inactive except for the patient upon whom pneumonectomy was done. This death is attributed to the resection and there was no other mortality in the thoracoplasty group.

Subcostal Plombage: There are some patients whose bilateral disease is such that control of the side of major disease alone offers little gain. In a few patients in whom bilateral resection was impossible bilateral permanent collapse was accomplished with plastic sponges. The attainment of collapse of diseased lung tissue without the deformity and impairment of pulmonary function which follow the conventional thoracoplasty has been attempted over the years using various materials in the extrapleural or subcostal (extraperiosteal) planes to maintain collapse. The poor results and numerous complications of these procedures have deterred their use.[3] More recently the use of nonreactive plastic sponge which becomes enmeshed with living fibrous tissue when implanted in the body has offered greater chance of success.[5] The blood supply, through the intercostal vessels, of lung tissue adherent to the parietal pleura is preserved when the chest wall is collapsed in the extraperiosteal plane. The production of lung collapse equivalent to a standard 7 rib thoracoplasty may be accomplished rapidly in one stage. Detailed studies have demonstrated that extrapleural or

subcostal collapse of diseased lung is followed by surprisingly little impairment of pulmonary function provided the intrapleural space is not entered.[4]

This fact plus the utility of the procedure for bilateral procedures make the operation particularly suitable for the patient with low respiratory reserve and bilateral upper lung disease. Extensive experiences reported in the Scandinavian literature indicate that when the sponge is placed in the space formed by denuding the ribs of their periosteum over the area to be collapsed the incidence of complications is low.[5] Webb has recently reported the use of subcostal plombage in 150 patients with only 3 infections.[8]

We have used the Polystan sponge and have limited its use to patients for whom no other surgical procedure of value seemed possible. Sixteen such procedures have been carried out on 10 patients. The upper rib cage is exposed through the usual thoracoplasty incision and the number and length of ribs necessary for adequate collapse are stripped of periosteum. Preservation of the periosteum on the outer surface of the rib insures its viability. The plastic sponge is placed beneath the ribs against the intercostal tissues and the space is drained with a catheter connected to a water seal bottle. This usually is removed in 48 hours.

One patient, 70 years old, hospitalized 4 years with unilateral fibrocavitary disease had been rejected repeatedly for operation because of his age and poor respiratory reserve. His recovery following plombage was uncomplicated and he has been discharged. Two of the other 3 patients on whom unilateral plombage has been done have been recommended for contralateral plombage also. One has not been convinced of the need of operation and the other, a chronic alcoholic, left against advice.

Bilateral plombage has been done on 6 patients. One had bilateral large cavities, considerable compensatory emphysema, absent chest expansion and a poker spine due to rheumatoid arthritis. This patient believes his exercise tolerance to be as good or better than it was preoperatively. Another man with severe asthma also experienced little reduction in exercise tolerance after bilateral plombage.

Six of these patients had negative sputum by concentrate and culture before operation but all were and had been on drug therapy for a long period. This leaves the significance of negative sputum open to question especially with roentgenographic evidence of cavitation. One of these sputum negative patients with bilateral plombage developed an acute spread of his disease after medical discharge, probably due to failure to restrict his activity. Another, the patient with severe asthma mentioned above developed spread of his disease and remains hospitalized. The remaining 4 patients with sputum negative at operation have been discharged as apparently inactive.

Of the 4 with sputum positive at operation none has been converted to negative. Three had bilateral procedures and this was recommended in the other who left against advice. Another left the hospital against advice after completion of bilateral procedures and the third remains hospitalized. The fourth patient, who had bilateral cavitation, developed a bronchial fistula and infection about the

left sided sponge 5 months after its insertion. In the interval, contralateral plombage had been done. The sponge was removed from the left side and a small area of erosion in the arch of the aorta was found. A massive hemorrhage a few hours later resulted in death. This was the only complication in the group.

The over-all results in this group of patients have been poor with only 5 reaching a status permitting regular discharge as apparently inactive and one of these returned with spread of his disease. The poor results, however, are attributed to the character of the disease rather than to the procedure used.

Resection: Although emphasis has been placed upon the collapse procedures many of the 136 patients subjected to resection during the same period of time have presented relatively poor operative risk. Twenty of these patients were over 50 years of age, the eldest 67 years old. Bilateral resections were required in 4 patients one of whom was 52 and a diabetic. Two patients have been subjected to repeat resections homolaterally. One 26 year old woman, a severe and labile diabetic, had resection of the middle lobe when her disease recurred following lower lobectomy elsewhere. Removal of the remaining upper lobe was done one year later because of recurrent disease which did not respond to various drugs. No significant disease was palpable or apparent on chest films at the time of the middle lobectomy. The left lung in this patient remains clear and she has been discharged. The other patient is a 52 year old Negro in whom, after long term therapy of extensive disease, segmental removal of residual cavitation was complicated by bronchial fistula and empyema. The remaining lung tissue was removed at a later operation.

Pneumonectomy is done only when some part of the lung cannot be salvaged. The need for such operation indicates advanced disease and probably rather poor resistance to the disease. As a result, pneumonectomy is attended by a mortality 3 to 5 times that of pulmonary resection of lesser extent for tuberculosis. Many of the 13 subjected to pneumonectomy had significant amounts of contralateral disease. Five of these patients were over 50 years of age. The one death in the

TABLE 1

Surgical procedures

(Nov. 1953–Jan. 1956)

	Patients	Age Range	White	Mortality
	Procedures	Average	Negro	
Thoracoplasty	25/52	19–60 (39)	4/21	0
Plombage	10/16	27–40 (44)	6/4	1
Resection	136/142	12–67 (36)	76/60	3
Total	170*/210		86/85	4

* One patient listed under both thoracoplasty and pneumonectomy

pneumonectomy group occurred in the patient mentioned above in the thoracoplasty group.

Two other deaths have occurred in the resection group. One occurred in a 26 year old Negro man 4 days following removal of all the right lung except the basal segments. The other was a 48 year Negro woman who died 14 days following removal of the upper and lower lobes on the right. Although a low respiratory reserve was likely a factor in each death neither is wholly attributed to this. It is of interest that the 170 patients were equally divided as to race but all of the deaths occurred in the Negro patients. The surgical procedures are summarized in Table 1.

SUMMARY

A significant and increasing number of patients with pulmonary tuberculosis are encountered in whom the application of surgical treatment must often be compromised due to factors which increase the individual surgical risk. Such factors as (1) the increasing incidence of pulmonary tuberculosis in older people (2) effective chemotherapy which permits survival with extensive disease residua of patients previously doomed, and (3) the significant number of patients with associated but unrelated pathologic processes necessarily indicate groups presenting increased surgical risk. In these groups the use of surgical measures less precise than excision offers considerable hope of salvage from a precarious balance of chronic invalidism. Experiences with patients presenting poor operative risk have been considered. The use of bilateral resection, thoracoplasty with and without cavitary drainage and subcostal plombage in selected cases has been discussed. The minimal disturbance of respiratory function in the last group has been striking. The results generally have been encouraging except in the group treated by subcostal plombage. Since these were mainly salvage procedures the successes warrant further trial of the procedure in similar patients in the future.

CONCLUSIONS

The problem of the poor-risk patient with pulmonary tuberculosis will be solved by earlier recognition of the disease, better and continued therapy, and closer attention to the patient's socioeconomic problems involved by the disease.

The occurrence of pulmonary tuberculosis in the older patient should be remembered if early diagnosis is to be made.

When pulmonary excision is deemed unfeasible, permanent collapse measures are worthy of trial. A prerequisite for success is a patient with reasonably good resistance to the disease and one who will adhere to the therapeutic regimen.

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RUPTURE OF THE SMALL INTESTINE THROUGH THE INTACT ABDOMINAL WALL WITHOUT ASSOCIATED INTRAPERITONEAL INJURY

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Rupture of the small intestine through the intact abdominal wall is perhaps more common than is generally recognized. Five patients were treated at the John Gaston Hospital, Memphis, Tennessee, between May 8, 1954, and April 23, 1955. A review of the cases of 21 patients who were treated at this hospital during the past 14 years (1941–1955) revealed interesting data which tends to indicate a better prognosis now for this type of injury than has previously been reported. Counseller and McCormack[3] in 1935 reviewed 1313 cases and reported a total mortality rate of 73.4 per cent and an operative mortality rate of 60.7 per cent. In 1938, Totten[13] reported a total mortality rate of 56 per cent and an operative mortality rate of 31 per cent. By 1951, according to several authors, the operative mortality rate was between 25–40 per cent (table 1).

The purpose of this article is to present data derived from 21 consecutive cases of ruptured small intestine caused by blunt trauma to the abdomen, and to stress the necessity for early surgical intervention in those patients who present findings which will be discussed in this paper. Delay in operation, one of the common causes of mortality, led Bosworth[1] in 1946, and Ficarra[4] in 1951, to emphasize the importance of early laparotomy.

The exact mechanism by which the small bowel is ruptured through the intact abdominal wall is not always apparent. Why these perforations occur with predilection in certain regions is still a matter of theory. Particularly dangerous are situations in which unsuspected flows are delivered to the abdomen. Under such conditions no reflex muscle splinting is evoked to prevent the bowel from being crushed against the rigid vertebral bodies. Also forces which tend to whiplash a segment of bowel can cause perforation at or near the points of attachment either by a shearing action or by limiting the movement of the bowel over body prominences such as vertebrae or the promontory of the sacrum.[4, 5, 7] It is not surprising that the majority of small bowel perforations are found either in the upper jejunum or lower ileum. Many of these will be found within the 24 inches distal to the ligament of Treitz, or the distal 24 inches of the ileum.[1, 4, 7, 11]

Bursting or crushing types of rupture are not infrequently found in patients with history of trauma to inguinal hernias.[11, 12] In general, the bursting type of perforation will be found to involve the antimesenteric section of the bowel whereas crushing or tearing injuries usually will involve the mesenteric border as well.[8] The internal trauma produced by a blow to the abdomen may be far out of proportion to the actual force sustained.[6] Most cases will reveal no evidence

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TABLE 1

Mortality rates

	Mortality	
	Operative	Total
1935 Counseller & McCormack	60.7	73.4
1938 Totten	31.0	56.0
1938 Meyer & Shapiro	30.0	—
1944 Hunt & Bowden	25.0	33.0
1948 Bosworth	29.3	34.5
1951 Jacobson & Carter	—	42.0
1951 Ficarra	—	44.0
1955 Duncan	10.5	19.0

Note: Associated intraperitoneal injuries were found in some of the series reported in this table; however, none were present in the group of 21 consecutive patients treated at this hospital.

of trauma to the abdominal wall itself. It is this group which challenges the diagnostic acumen and skill of the clinician.

DIAGNOSIS

Despite the serious insult of a perforated small intestine, the early signs and symptoms may appear insignificant; consequently, the patient may not seek medical attention for several hours. The initial trauma usually is followed by nausea, vomiting, and vague abdominal pain, and if a perforation is present, the pain will become progressively more severe. Continuous, persistent pain in patients who have had abdominal trauma probably is the most important symptom.[3] Abdominal rigidity is, of course, one of the most significant physical findings. In most cases adynamic ileus occurs early, and it is followed later by a distended tympanitic, silent abdomen.[13] The pulse rate usually is accelerated and the leukocyte count is most commonly elevated. Although no physical sign is pathognomonic, the roentgenologic demonstration of free air within the peritoneal cavity or in the retroperitoneal area is often diagnostic. Pneumoperitoneum is more frequently associated with high jejunal perforation than with perforation of the lower ileum, because the latter normally is devoid of gas.

Twenty of the 21 patients in this group had a satisfactory roentgenographic examination. In none of the patients with perforation of the ileum was a pneumoperitoneum demonstrated, but 9 of the 14 patients (64 per cent) with ruptured jejunum did have this very significant finding. Sixty per cent of the 20 patients who were examined by roentgenogram had either pneumoperitoneum or films which were otherwise diagnostic.

For the demonstration of free air within the peritoneal cavity, the posteroanterior upright chest film is preferred since very small amounts of air can be readily detected (fig. 1). If the patient is too ill to stand, the left lateral decubitus position usually will display the air between the right lobe of the liver and the diaphragm (fig. 2). The abdominal roentgenogram, which is taken with the

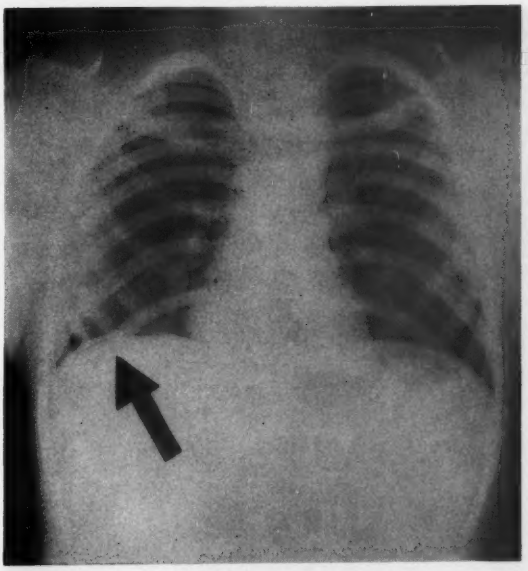

FIG. 1. Pneumoperitoneum (Case 18) demonstrated on posteroanterior upright chest roentgenogram which shows a very small amount of free air beneath the right leaf of the diaphragm.

FIG. 2. Pneumoperitoneum (Case 15) left lateral decubitus film of the abdomen with free air beneath the diaphragm and right lobe of the liver. This patient was too ill for the upright posteroanterior chest film.

FIG. 3. Supine abdominal film displaying perirenal emphysema in Case 20. This patient had retroperitoneal rupture of second portion of the duodenum.

patient in a supine position, occasionally will denote free fluid within the peritoneal cavity as evidenced by an over-all increased density and, in some patients, may show air trapped beneath the transverse mesocolon opposite the second lumbar vertebra.[10] If possible, it is recommended that both the abdomen and chest be examined by roentgenogram. The abdominal film, in addition to giving information concerning free fluid within the peritoneal cavity, will provide the answer in many instances of retroperitoneal rupture of the duodenum. The findings in such cases are retroperitoneal emphysema and usually the outlining of the right kidney by a perinephritic air collection (fig. 3).

Since a roentgenogram will confirm the diagnosis of a ruptured hollow viscus in only 50 to 60 per cent of the patients, it is obvious that a negative roentgenogram is of no value in arriving at the correct diagnosis. Although the introduction of air into the bowel either by nasogastric or colon tube has been recommended as a diagnostic aid,[2] it is believed that this procedure is not without hazard.

Other previously described physical findings such as absence of liver dullness to percussion, and the localization of pain to the left of the umbilicus[3, 14] were not reliable in this group.

One finding of possible prognostic significance was the presence or absence of shock. All of the patients, except one, who were in shock at the time of admission died regardless of therapy. This includes one patient who died on the way to the

ward from the emergency room. There may be an early transient period of shock immediately following the injury, but patients at time of admission usually are not in profound shock.[7] As recently as 1938, surgery after 12 hours was considered almost 100 per cent fatal. However, 6 patients in this group were not seen until at least 12 hours after trauma and, indeed, others were not admitted until 3 days, 5 days, and another, 6 weeks after trauma.[11] These were operated upon and survived. In each of these instances the intestine probably had been devitalized at time of trauma and the injury to the wall resulted in a delayed rupture.

Cases no. 20 and no. 21 are of special interest and are briefly presented.

CASE REPORTS

Case 20. H.L.H., a 40 year old Negro man, was first seen in the emergency room of the John Gaston Hospital 18 hours prior to admission for suture of laceration above the left eye. Twelve hours before admission, while in the city jail, he began to complain of severe, right upper quadrant pain. This pain became progressively more severe and was accompanied by nausea and vomiting. On admission his temperature was 101 F., pulse 90, and blood pressure 130/80. Chest examination was negative. There was marked right upper quadrant abdominal tenderness with rebound tenderness referred to this area.

Laboratory findings revealed the hematocrit was 41 vol. per cent, the leukocyte count was 11,200 per cu. mm. and the urinalysis was negative. Roentgenogram showed retroperitoneal collection of air around the right kidney.

At laparotomy a 1 inch transverse laceration was found in the retroperitoneal portion of the duodenum. This laceration, which was directly opposite the ampulla of Vater, was closed without difficulty. Except for drainage of a retroperitoneal abscess on the eighteenth postoperative day, the patient made a satisfactory recovery.

Case 21. H.J., a 16 year old Negro boy, had been struck in the abdomen by a tail-gate of a truck 6 weeks prior to admission to the hospital. For 1 week following the accident, he complained of epigastric pain. Two days before admission to the hospital, the epigastric pain recurred and was followed by nausea and vomiting. All of these symptoms increased in severity during the 48 hours before admission. When he was examined on the ward his temperature was 101.6 F., pulse 100, and blood pressure 110/70. The abdomen was moderately distended, tympanitic, very tender and rigid.

Laboratory examination revealed the leukocyte count was 16,750 per cu. mm., the hemoglobin was 14 Gm. per cent and the urinalysis was negative except for 5–10 red blood cells per high power field. The roentgenogram showed pneumoperitoneum (free air beneath right diaphragm).

At laparotomy the jejunum was found to have been completely transected 12–14 inches distal to the ligament of Treitz, but it had been incorporated by the greater omentum in such a fashion that the continuity of the lumen had been preserved until it had reperforated. This portion of the bowel was resected and an end to end anastomosis performed. His postoperative course was uneventful.

TREATMENT

Immediately after the diagnosis was established nasogastric suction was instituted. This was continued postoperatively until intestinal motility had returned. Other supportive therapeutic measures which included the administration of blood as necessary, fluids, electrolytes, parenteral vitamins, and antibiotics were given in order to prepare the patient for surgery as rapidly as possible.

The definitive surgical treatment in this group was excision of devitalized intestinal wall with primary closure of the defect, or resection of the involved segment and primary anastomosis. External drainage from the site of rupture was rarely employed following closure, and no type of tube enterostomy proximal to the anastomosis was utilized in these 19 patients.[8]

MORTALITY

All but 2 of this group of 21 patients were treated surgically. One patient died en route to the ward from the emergency room. The other patient who was not operated upon did not respond satisfactorily to vigorous preoperative preparatory measures. There were 4 deaths giving a total mortality (including the dead on arrival) of 19 per cent. The operative mortality, however, was 10.5 per cent since only 2 of the 19 who underwent surgery did not survive.

DISCUSSION

Traumatic rupture of the small intestine through the intact abdominal wall is by no means rare. Serious injuries have been found to follow minor trauma. Delayed perforations may occur in a segment of bowel that was devitalized but not ruptured at time of injury. Instances in which a patient with a perforation found at surgery 5 days to 6 weeks after trauma would strongly indicate that this had occurred.

A person with acute perforation most commonly presents himself within 4 to 6 hours after the injury. The most significant physical findings are persistent, progressively increasing abdominal pain, nausea, vomiting and spasm of abdominal muscles. Such a patient usually is not in profound shock.

Roentgenologic examination may be diagnostic of a ruptured hollow viscus in 50–60 per cent of cases if both flat film of the abdomen and upright posteroanterior chest films are made. Although roentgenologic examination may not give positive evidence of a ruptured viscus in 40–50 per cent of cases, patients who have had blunt trauma to the abdomen and who have nausea, vomiting, abdominal muscle spasm, and other findings compatible with an acute condition within the abdomen should have laparotomy as soon as they can be properly prepared. The risk of surgical exploration is small compared to the nonoperative treatment for this condition.

In cases of perforation of the duodenum it is necessary to examine the retroperitoneal portion as well as the anterior surface at the site of rupture. One of the postoperative deaths in this group of 19 patients can at least be partially attributed to the overlooking of a posterior perforation. This patient died 7 hours postoperatively.

Although no case of massive intraperitoneal hemorrhage was found in this group, the presence of a large amount of blood makes mandatory the careful examination of the liver and spleen to exclude rupture of these organs.

The steady decrease in mortality rate since 1935 indubitably can be attributed to better anesthesia, the modern blood bank, antibiotics, chemotherapeutic agents, and a better understanding of fluid and electrolyte problems by the clinician.

SUMMARY

A review of 21 consecutive cases of ruptured small intestine through the intact abdominal wall without associated intraperitoneal injury is presented.

The mechanism of injury and pertinent physical and laboratory findings are discussed.

The importance of early diagnosis and aggressive supportive therapy followed by closure of the perforation without delay is emphasized.

Two brief case reports are given, one involving rupture of the retroperitoneal portion of the duodenum and the other involving a delayed rupture of a completely transected jejunum.

The sharp decline in both total and operative mortality in the past 20 years is shown and can be attributed to early corrective surgery, the modern blood bank, antibiotics, chemotherapeutic agents, a clinical understanding of fluid and electrolyte balance, and modern anesthetic technics.

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SUCCESSFUL EMBRYONIC PARATHYROID TISSUE TRANSPLANT FOR THE TREATMENT OF INTRACTABLE POSTOPERATIVE PARATHYROID TETANY

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The onset of clinical tetany following operations upon the thyroid gland is one of the most tragic and unfortunate complications that can confront the practicing surgeon.

Bell and Bartels[1] reported 58 cases following 2000 thyroid operations or an incidence of 3.0 per cent. This approximate percentage holds true for other clinics following operations for thyrotoxicosis, carcinoma, or thyroiditis. In recurrent thyroidectomies the incidence is somewhat higher.

Fortunately, most cases of parathyroid dysfunction following surgery are transient and present no particular problem in management. However, it is generally believed that tetany persisting a year following surgery will most always be permanent. Management of these cases of permanent parathyroid deficiency often is difficult and costly and may be complicated by the formation of cataracts, convulsive episodes, mental deterioration, and psychosis.

After having had experience in the difficult medical management of one of these patients with permanent and severe tetany, the authors believe that the answer lies in a parathyroid transplant. Parathyroid gland transplant was accomplished experimentally in 1929 by Cattell[2], and again in 1934 by Stone and Owings.[7] These authors used the tissue culture method of preparing the graft for transplant.

Houghton and Klassen[3] reported a transplant to the axilla was successful in the symptomatic relief of a patient for 6 months.

In April, 1954 Sterling and Goldsmith[6] in Philadelphia reported a successful total transplantation of the thyroid gland using vascular anastomosis. Since that report Dr. Sterling has had 2 more successful cases, making a total of 3.

We were not aware of Dr. Sterling's work at the time we considered a transplant on our patient.

Acting on the kind advice of Dr. Edward T. Krementz[4] of Tulane University, we decided to attempt an homologous free graft using fetal embryonic parathyroid tissue—the recipient area was to be the anterior abdominal rectus muscle. Dr. Krementz pointed out to us that in the experimental animal an homologous take of embryonic tissue would be more likely to succeed if the anterior chamber of the eye or the brain could be used as a recipient area; but, of course, these were impractical in the human being.

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CASE REPORT

The patient, a 29 year old white woman, was admitted to The McLeod Infirmary on Jan. 3, 1951 with a diagnosis of thyrotoxicosis, moderately severe. For several months prior to admission she had been treated by the Medical Service as an out-patient for hyperthyroidism and had not responded adequately to propylthiouracil. Subtotal thyroidectomy had been recommended and accepted by the patient.

The past history was noncontributory except for one hospital admission several years previously at which time the discharge diagnosis was psychoneurosis. She also had been treated with a dilatation and curettage and radium 10 years ago for metrorrhagia and menorrhagia, and since this time she has menstruated every 2 weeks, regularly.

Physical examination at time of admission disclosed a well developed and fairly well nourished white women, age 29, and weighing 107 pounds. Her pulse was 100, blood pressure 120/72. The thyroid gland was symetrically enlarged. No distinct nodules were felt. The blood picture was normal and urine negative. She had been on Lugol's solution for about 2 weeks prior to admission.

On Jan. 6, under intratracheal general anaesthesia a bilateral subtotal thyroidectomy was carried out. Nothing unusual was noted at the time of surgery except no parathyroid glands were seen or recognized as such. Her immediate postoperative period was normal until about 24 hours following surgery, at which time there was an explosive onset of tetany. This was apparently preceded several hours by some tingling of the hands and around the mouth. Carpopedal spasm and evidence of neuromuscular irritability (Trousseau's, Chvostek's and Erb's signs) was present. A feeling of restriction of freedom of inspiration and expiration was noted. Calcium gluconate, 10 cc. of a 10 per cent solution, was immediately given intravenously. The response was dramatic. It was necessary to repeat this dosage about every 8 hours, however, until she could be stabilized by oral medication. She was fairly well controlled in a few days on calcium lactate, 30 gr. four times a day; Hytakerol (dihydrotachysterol), 1 cc. four times a day, and ammonium chloride, 15 gr. four times a day. Blood serum calcium on one occasion was 8.8 mg. per cent. She was discharged from the hospital on Jan. 20, 1951—17 days after admission and fairly well controlled on the oral medication as outlined.

The surgical specimens were carefully reviewed by the pathologist as soon as hypoparathyroidism became evident. There was one parathyroid body attached to the capsule of one lobe and one parathyroid body enfolded beneath the surface of the gland in the same lobe. A careful search and multiple sections through both resected lobes failed to reveal more than the two parathyroid bodies which were identified by microscopic sections.

The next 2 years were a series of nightmares for the authors and other physicians who helped with her case. From the time of her discharge in January 1951 until February 1954, she was readmitted to the hospital 11 times. This represented a total of 363 days of hospitalization before improvement became evident. During this time she was having frequent attacks of tetany and could not tolerate her oral medication which seemed to cause nausea, vomiting, and diarrhea. When oral medication could not be tolerated she was given glucose intravenously which contained 20 cc. of 10 per cent calcium gluconate and supplementary vitamins B and C several times a day. Parathyroid hormone in the form of Parathormone was used liberally, but we could not see much benefit from its use. During this period she complained more and more of vague skeletal pain, which required frequent doses of narcotics and barbiturates. Her diet, when she could tolerate one, was kept low in phosphorus. Blood calcium determinates were not enlightening—they varied from 5.5 mg. per cent to 11 mg. per cent. The serum phosphate ranged from 2.4 mg. per cent to 3.5 mg. per cent. The Sulkowitz test for calcium in the urine almost constantly showed 2+ to 4+. It was evident that she was rapidly deteriorating both mentally and physically, although as yet no signs of cataracts or skeletal abnormalities were noted. Medication by mouth had been juggled in every conceivable fashion, and except for about one-third of this time when she was apparently under control, her general course was very unsatisfactory.

In August 1953, Dr. Kenneth Barrett, one of the interns on the Medical Service at that time, volunteered to write to Dr. Edward T. Krementz of Tulane University, whom he knew to be interested in this problem. Dr. Krementz[4] advised us by letter to attempt a transplant from a viable fetus of about 3–4 months gestation and use the abdominal rectus muscle as a recipient area. Accordingly, the Obstetrical Department was alerted to notify us when a therapeutic abortion was to be done. About 1 month later on September 6, the Obstetrical Department notified us that a therapeutic abortion was to be done that day. Since our patient was already in the hospital, she was taken to the operating room at the time of the uterotomy and the recipient area prepared. Under local anaesthesia this fetal tissue was implanted into the right rectus muscle about 2 inches below the umbilicus. No attempt was made to use a donor with the same blood type as the recipient.

About 5 months later it was noted that the patient's clinical course had begun to improve constantly but slowly. She ceased to have the attacks of tetany and finally all oral medication was stopped. She was discharged from the hospital on Feb. 9, 1954 on no medication. Her next admission was 10 months later at which time she had some mild tingling sensation during the course of an illness of influenza. At this time she was maintained on 1 cc. of Hytakerol a day and no calcium by mouth. During this interval in the hospital it was noted that the serum calcium levels were consistently higher—averaging 9.65 to 15.5 mg. per cent.

More recently on the advice of Dr. Sterling we have taken her off Hytakerol since he believed that it might suppress the functioning parathyroid tissue. She still has some tingling at the time of her menses which, as stated, occur every 2 weeks. Her only medication at the present time is calcium lactate, 15 gr. three times a day. In the meantime she has gained back to her normal weight and looks healthy both mentally and physically. She works at her job regularly.

One most interesting fact is that the color of her hair, which was auburn, has changed to a very dark brown, almost black, since the transplant.

SUMMARY

One case of parathyroid homologous transplant that seems to have been successful has been presented. The patient suffered from intractable hypoparathyroidism that was surgically produced for 3 years. During this period she spent approximately one third of the time in the hospital. She has been almost asymptomatic for the past 2 years aside from several minor episodes, and is at the present time controlled on only minimal doses of oral calcium.

We believe that we were extremely fortunate in getting a take by the method presented. In view of the published works of Dr. Julian Sterling,[5] who reported 3 successful takes using the vascular anastomosis method, we believe his method to be the one of choice in spite of the technical difficulties that must be overcome.

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INTRAVENOUS CHOLECYSTOGRAPHY AND CHOLANGIOGRAPHY USING CHOLOGRAFIN

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The gallbladder and the bile ducts usually are not seen on plain roentgenograms of the abdomen because their contents are not dense enough to cast a shadow. To facilitate the visualization of these structures intravenous cholecystography was developed by Graham and Cole in 1924.[3] Oral contrast preparations of high iodine content were subsequently introduced producing good gallbladder filling, but until oral Telepaque (iodopanoic acid) came into usage the biliary ducts were never consistently visualized. After cholecystectomy, cholangiograms using Telepaque have been reported in 72 per cent of patients by one group of investigators,[8] but others have not achieved such good results. The only other reliable method of cholangiography is surgery with the direct injection of radiopaque materials into the cystic or common ducts.

In 1953 German investigators,[1, 2, 4, 5] reported their clinical and roentgenographic experiences with a new intravenous bile contrast compound called Biligrafin (sodium iodipamide), now known in this country as Cholografin (Squibb). This contrast media has a very high degree of radiopacity due to its high iodine content (64.32 per cent). After its administration satisfactory cholangiograms and cholecystograms are obtained in a very high percentage of the cases, and in cholecystectomized patients the duct visualization remains good. Cholografin is much less toxic than the older intravenous preparations used for gallbladder examination. Approximately 10 per cent of the material is normally excreted by the urinary tract.

Preparation and technic: Gastrointestinal cleansing and dehydration have been found to be very important in the preparation of the patient. Routine skin testing and slow injection of the contrast material over a period of 10 minutes has kept reactions to a minimum. Unpleasant side reactions can be controlled by the administration of anti-histamine, ephedrine and oxygen.

Optimum visualization of the hepatic and common bile ducts occurs 20 to 40 minutes after completion of the injection. When the gallbladder has not been removed it begins to fill within an hour with the best concentration in 2 to 2½ hours. If a gallbladder image is not obtained the patient is continued on a fat free diet and delayed films which may prove to be of diagnostic value are taken 18 to 20 hours after injection. Paregoric and morphine sulfate have definitely improved the radiographic appearance of the duct system. Body section radiography is used to help exclude confusion produced by gas in the intestinal tract or contrast material in the duodenum or kidney.

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FIG. 1. L. R., a 75 year old white woman with a long history of gallbladder disease. Oral cholecystogram showed no function and no visualization of the biliary system. Intravenous cholografin demonstrates marked dilatation of the intrahepatic, common hepatic and common bile ducts. There is a large nonopaque stone in the common hepatic duct and another stone of similar size in the common bile duct. The gallbladder is faintly visualized and it too contains stones. The gallbladder containing stones was removed, and the common duct explored with removal of the two stones.

Indications: Oral Telepaque studies may demonstrate 60 to 70 per cent[6, 7] of the biliary ducts on routine cholecystographic examinations. When the oral method has failed, visualization of the ducts may be accomplished by the intravenous method. Unsuspected stones may be discovered and the caliber and course of the ducts may be determined (fig. 1). Many surgeons are now requesting such preoperative studies before cholecystectomy because they need a thorough knowledge of the entire biliary system before surgery so as to avoid trouble later on (fig. 2). Fifteen per cent of the examinations in this series were for this purpose.

Cholografin will show filling of the gallbladder in approximately 65 per cent of the cases judged as nonfunctioning by the oral route (fig. 3). It must be pointed out, however, that about 5 per cent of nonfunctioning gallbladders are not actually pathologic and the nonvisualization may be due to other factors. Such factors which may be readily overcome by the intravenous method would include incomplete absorption from the gastrointestinal tract in patients with diarrhea, or other intestinal disturbance, inability to retain the contrast media because of severe nausea or vomiting, or simply failure of the patients to take the tablets.

Unfortunately, in some postcholecystectomy patients, symptoms may persist or there may be a recurrence of symptoms. Because Cholografin shows the biliary

FIG. 2. A.R., a 60 year old white women with a history of biliary colic. Intravenous study was requested to determine if stones were present in the common duct. Good filling of the ducts and gallbladder is demonstrated. The common duct is normal. All the gallstones are in the gallbladder.

FIG. 3a. J.B., a 36 year old white man. Two areas of calcification are seen in the right upper quadrant. Oral cholecystogram showed no visualization of the gallbladder or ducts.

FIG. 3b. J.B., a 36 year old white man. Cholografin shows filling of a small contracted gallbladder containing stones. There is a single stone impacted in the neck of the gallbladder. The common duct appears normal.

ducts with such a high degree of accuracy, it was natural therefore, that it would find great use in this particular group of patients (fig. 4). Stones may be inadvertently left in the intrahepatic or common bile ducts. Inflammation and trauma to the common duct may result in fibrosis, stenosis and stricture with resultant dilatation (fig. 5). Inflammation, stone formation or neuroma may develop in a cystic duct remnant or in the residual stump of the gallbladder (fig. 6).

In some instances of acute abdominal distress the diagnosis may not be evident and the intravenous method may be of considerable help in arriving at an early diagnosis. An absent or faint gallbladder shadow may be present with acute cholecystitis or acute pancreatitis using Cholografin.

Intrinsic tumor of the gallbladder or the ducts may be discovered. Extrinsic tumor is more common, especially metastatic nodes or primary tumor of the pancreas or duodenum and may show impingement or displacement of the visualized biliary system. Patients with carcinoma of the head of the pancreas usually are jaundiced and Cholografin is of little help as the common duct is seldom visualized. No involvement of the common duct was demonstrated in one patient with a very large sarcoma of the duodenum (fig. 7).

There has been an occasional request for follow-up studies of the ducts several months after cholecystectomy in asymptomatic patients in which stones were removed at the time of surgery. Assurance that all the stones have been removed is gratifying information to the surgeon. Such follow-up studies might also help in determining whether or not compensatory dilatation of the ducts occurs after surgery.

FIG. 4. D.K., a 37 year old white woman, was a postcholecystectomy patient. Intravenous cholografin demonstrates good filling of the cystic, intrahepatic, common hepatic and common bile ducts. The media has passed readily into the duodenum outlining the mucosa of the second and third portions. Even with good visualization the terminal portion of the common duct is not well visualized. The patient had no further surgery.

FIG. 5. N.W., a 58 year old white woman whose gallbladder had been removed 2 years prior to admission and who had had recurring attacks of colicky pain for the past year. Cholografin demonstrates a dilated common duct. No stones can be seen but at operation a small shot-like stone was found impacted near the sphincter of Oddi.

FIG. 6. A 53 year old white man, 2 years postcholecystectomy. Developed localized pain in the right upper quadrant radiating to the back accompanied by nausea. Cholografin demonstrated a normal common bile duct and a very short remnant of the cystic duct. At surgery a neuroma of the cystic duct stump was removed.

Results: There were 45 patients examined by the intravenous method who had had no previous gallbladder or duct surgery. Gallbladder visualization was accomplished in 31 patients and satisfactory duct visualization in 32. Of 29 patients who had nonfunctioning gallbladders by oral studies, 19 showed gallbladder and common duct filling. In this latter group, stones were demonstrated in the gallbladder in 6 patients, in the cystic duct in 1 patient, and in the common bile duct in 3 patients. Stones were demonstrated in the gallbladder of 4 other patients. Examination of 3 patients with acute cholecystitis produced no filling of the biliary system in 1 patient but in 2 patients there was faint visualization of the gallbladder and good common duct filling. In acute pancreatitis poor visualization of the gallbladder and good visualization of the biliary ducts was accomplished in 1 patient, but in another no filling of the entire biliary tract was obtained.

Sixty postcholecystectomy patients were examined using Cholografin. The hepatic and common bile ducts were visualized in 57 patients and in 88 per cent

FIG. 7. C.K., a 63 year old white man with an extensive sarcoma involving the second portion of the duodenum. Involvement of the ampulla of Vater was suspected but this was ruled out by cholografin study.

of these the visualization was good. The common bile duct was considered dilated when it measured more than 1 centimeter in diameter. Thirteen patients had dilated common bile ducts and 4 of these patients had subsequent surgery. No stones were demonstrated radiographically in this group; however, a small stone was found inpacted near the sphincter of Oddi in a patient at surgery. One patient had acute pancreatitis and a slightly dilated common duct was demonstrated. The cystic duct was visualized in 26 patients and because of symptoms localized to this area, 3 patients required subsequent surgery to relieve symptoms.

Failures and contraindications: Cholografin has been administered to 11 patients with jaundice and in only one instance was the bile duct visualized. Following this experience the use of this compound was discontinued in jaundiced patients in our clinic. In 3 patients, for no apparent reason, no shadows of the biliary system were obtained. Lack of proper dehydration and unrecognized liver disease may have been etiologic factors. In some instances, in spite of special

technic, gas and feces interfered with the radiologic image. The lower end of the common duct usually is very difficult to examine because of superimposition of duodenal mucosal folds and small stones can be easily missed. A history of hypersensitivity of the patient to iodine, a positive skin test, liver disease, severe renal disease, hyperthyroidism, and jaundice constitute contraindications.

SUMMARY

A brief history describing the development of contrast media used in cholecystography and cholangiography has been presented. Clinical experience and results in 105 patients with the latest of these media, Cholografin, has been given. The contraindications, indications and failures with this compound have been discussed. Because of its great density, no concentration is required in the gallbladder and thus the media serves as a filling procedure rather than as a function test. Cholografin serves as a valuable adjunct to the oral methods now in use and in certain instances entirely supplants the oral methods in diagnostic problems of the biliary system.

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EDITORIAL

NOT FOR OURSELVES ALONE

The good Lord gives specific gifts to each and every person at birth. We would learn that this is also true of every other living thing if only we had an intelligent interpreter for each form of life. Such gifts signify that there is a purpose for the existence of living things. There is also a purpose for the existence of inanimate or material things, for without them, there could be no life.

We who delight in calling ourselves "mankind"—at times with tongue in cheek and often, in this country, with the feeling that the term includes only those who speak the English language—have much to learn about the purpose of our existence. We first must learn about ourselves. Socrates put it in two words, "Know thyself." To this some might reply, "That is a lifetime study in itself and leaves no time for action." My reply, however, is that knowledge of ourselves leads eventually to but one conclusion, namely that we do not exist for ourselves alone and that, consequently, we should lead a devoted, purposeful life in a chosen field of worth-while human endeavor.

We must know something about other people's problems. A person must be mentally sick if he can exist on earth but live so high in the clouds that he is unaware of what is going on about him. We are in no Utopia; rather, we are in a world of many faults, and not all of them belong to the other person.

In the United States, we are blessed with more of the material things of life than are the people of any other country. We as a group, as well as individuals, are gradually coming to the idea that our concept of life in terms of the freedoms as we know them should be the right and privilege of all mankind. We would not, however, thrust this concept upon other peoples who are separated geographically from us and have different creeds, any more than we would want their concepts thrust upon us.

The path of true progress in international affairs must be trod in an orderly manner, step by step, both at home and abroad. Progress stems not primarily from huge monetary gifts but rather from the education of native persons as teachers who in turn may educate their own peoples in the true values of life and living as they may be adapted to their own souls and soil. I would pick medicine, agriculture and engineering as the three principal fields for education and endeavor in those countries less fortunate than ours. These fields could contribute best to international co-operation and understanding, which are the watchwords of peace.

The day of the individual isolationist will never be over, but the day of group isolationism is past.

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BOOK REVIEWS

The Editors of The American Surgeon will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.

Fluid Balance Handbook for Practitioners. By WM. D. SNIVELEY, JR., M.D., A.A.G.P. MICHAEL J. SWEENEY, M.D., A.A.P. Both authors are Attending Physicians, Evansville Child Health Clinics, Staff Member St. Mary's Hospital, Med. Dept., Mead Johnson & Company. Cloth binding, illustrated, 326 pages. Springfield, Illinois, Charles C. Thomas, Publisher. Price $6.75.

This is an interestingly written volume. The approach is different—here is a mixture of the sayings of Oliver Wendell Holmes, Joseph Conrad, The Bible, Sir William Osler, as well as many others sprinkled in to add a touch of explanation and readability to an otherwise drab subject. In their introduction, the authors state that they have incorporated various memory and visual aids which have "proved immensely useful in teaching this subject to practitioners".

The book is written in simple easy form. This first part of the book is devoted to an explanation of the theory of fluid balance and imbalance. The theory here is not heavy as in Gamble or Moyer's texts, but is almost conversational. The latter part is devoted to clinical history showing certain common conditions, the treatment used, and the results obtained. The fluid balance diagnosis and therapy is presented at the end of each chapter, and at first thought it would seem most eventualities and conditions have been covered; at least, the authors feel so for they said in effect if you don't understand, then you should just reread that chapter. I fear fluid and electrolyte balance is not so easily settled.

It seems the name of "Handbook" is not quite fitting, for a handbook is one that is full of nothing but pertinent information, but this book is a nice review of the subject. Other more detailed texts will be needed by the student of fluid and electrolyte problems.

A. H. LETTON, M.D.

Anatomy for Surgeons, Volume II. By W. HENRY HOLLINGSHEAD, PH.D., Professor of Anatomy, Mayo Foundation, University of Minnesota, Head of the Section of Anatomy of the Mayo Clinic, Rochester, Minnesota. Clothbound, illustrated, 934 pages. Published by the Medical Book Department of Harper and Brothers, 49 East 33rd Street, New York 16, N. Y.

This anatomy is written especially for surgeons and is not intended to be a complete, descriptive volume, but rather is a practical description of the anatomy along with its interpretation so as to be of greater value to the surgeon who deals with this anatomy at the operating table. The book is said to be a direct outcome of the author's experience as a teacher of Graduate Fellows of the Mayo Clinic and Foundation.

After the book was written, the various chapters were given to various members of the surgical staff of the Mayo Clinic who criticized them as to content and applicability to the surgery of the area. Such suggestions as were made were incorporated after necessary consultation.

The fundamental anatomy, especially that not easily seen at the operating table, is stressed so that one may use this as a foundation in dealing with the area or part. The two goals of the author in preparing the text were (1) to provide not only a review of the broad basic anatomy, but also a discussion of the details upon which the surgeon bases his work, and (2) provide for more mature surgeons those details which are occasionally needed for reference as well as for a refresher.

This book is a good, valuable, practical book for either the young or mature surgeon, written by and corrected by people whose everyday life is majorly affected by the teaching and use of anatomy.

A. H. LETTON, M.D.

Plastic Repair of Genito-Urinary Defects. By GEORGE BANKOFF, M.D., D.CH., F.R.C.S. Ed., F.R.F.P.S. Formerly Associate Professor of Surgery (Plastic and Reconstructive Surgery), Georgetown University Medical School, Washington, D. C. Plastic Surgeon, Gallinger Municipal Hospital, Washington, D. C. Formerly E.M.S. Surgeon, St. John and St. Elizabeth Hospital, London. Assistant Surgeon, University Hospital, Bonn. Chief Surgeon, Plastic Department, George Eastman Institute, Rome. The Philosophical Library, New York. Price $17.50.

Had the author used a less loose definition of "Genito-Urinary Defects" he may have produced a useful book. As it is, he has produced a volume which only partially covers the repair of Genito-Urinary Defects. A considerable portion of the book is devoted to pure urology and gynecology, the details of which can better be found elsewhere. In committing the latter offense the author has sacrificed clarity for brevity. In the portion of the book implied by the title, some of the more recent procedures for the repair of hypospadias are not included. In no sense can this book be regarded as an "Atlas". The illustrations are of inferior quality.

This book adds nothing to the medical literature. The reviewer cannot see how it could be of use to an experienced plastic surgeon and as there is little effort made by the author to point out the inherent defects in each operation, the novice had better beware of its use as a reference book.

CHARLES EBERHART, M.D.

INDEX TO VOLUME XXII

Book Reviews, Editorials and manuscripts on Surgical Technic are grouped together and are indexed under those headings in alphabetic order.

Abdominal
  aortic aneurysms, 623, 1182
  incisional hernias, 217
  roentgenogram interpretation, 249
  wounds, wire closure, 746
Abdominal Pregnancy: Report of Four Cases, A. B. Wolfe, V. W. Brabham, and R. C. Horger, 1029
Abronchiectatic Bronchiectasis: The Recognition of Surgical Pneumonitis, R. E. MacQuigg, 465
Accidental Head Injuries in Occupants of Crash Motor Vehicles: A Report of Two Hundred and Seventy-Three Cases, J. Kulowski, 528
Accuracy in the Diagnosis of Jaundice with Particular Reference to the Intravenous Galactose Tolerance Test, C. A. Macgregor, and G. A. Munro, 657
Acute Appendicitis in the Elderly Male, W. J. Gillesby, and C. M. O'Reilly, 1186
Acute Cholecystitis: An Analysis of Sixty-One Cases, W. C. Bartlett, 119
Acute Emphysematous Cholecystitis, P. R. Yedinak, 664
Acute Gastric Dilatation, W. J. Gillesby, and J. R. Wheeler, 1154
Acute Pancreatitis with Necrosis and Perforation of Common Bile Duct: Case Report, D. E. Butler, and R. Eichhorn, 512
Acute Perforated Peptic Ulcer—An Analysis of Eighty-Seven Cases, J. H. Growdon, and R. M. Stainton, 278
Acute Perforated Peptic Ulcers, B. Lewis, and J. E. Hamilton, 204
Acute Spinal Injuries, B. W. Cannon, 1108
Adenomatous Polyps of the Large Bowel: Their Significance to the Surgeon, J. E. Thompson, and R. L. Pinck, 964
Adrenal
  cortex, 56
  cortical insufficiency, 1117
  glands, surgery, 904
Adriani, J.: Anesthesia for the Aged and Poor-Risk Patient, 1023
Advances in Technical Aspects of Biliary Tract Surgery, H. Mahorner, 615
Alley, R.: See Floyd, J. B., Jr.
Allison's principle, 668
Altemeier, W. A., and Cole W.: The Future of Antibiotic Treatment in Surgery (Editorial), 821
Amputation
  extremity, 696
  poor-risk patients, 1023
Ancient Anodynes: A History of Anesthesia Prior to 1750 (Historical), R. C. Groth, 345
Anesthesia
  careless, 1038
  prior to 1750, 345
  refrigeration, 998
Anesthesia for the Aged and Poor-Risk Patient, J. Adriani, 1023
Anesthetic Complications in Pediatric Thoracic Surgical Patients, R. P. Bergner, 187
Aneurysms
  abdominal aortic, 623
  aortic, 1043, 1182
  intracranial, 100
Anorectal Complications of Pregnancy, W. H. Gerwig, Jr., 549
Anterior Sacral Meningocele, R. S. Silvis, L. R. Riddle, and G. G. Clark, 554
Antibiotic treatment in surgery, 821
Anus
  basal cell carcinoma, 886
  carcinoma, 641
  imperforate, 1112
Aorta
  coarctation, 1168
  homologous grafting, 428
Aortic
  aneurysms, 623, 1182
  aneurysms, surgical treatment, 1043
Appendicitis, in elderly male, 1186
Application of Neck Gland Resections, R. W. Buxton, 16
Arterial
  homografting, 1163
  prostheses, 1134
Arteriosclerotic abdominal aortic aneurysms, 623

Artery
anterior communicating, 100
grafts, nylon, 1174
mammary, delayed hemothorax, 135
Atrial septal defects, 1058
Austin, D. J., and Thompson, J. E.: Treatment of Peripheral Arteriosclerotic Disease by Arterial Homografting, 1163
Autografts, venous, 455

Bacterial Factors in Strangulation Obstruction: Review of Experimental Work, I. Cohn, Jr., 836
Baker, J. P.: See Fitchett, C. W.
Blakemore, A. H.: Progress in the Development of Arterial Prostheses (Editorial), 1134
Barrett, C. M., and Sanger, E. L.: Radiation Hazards in the Practice of Surgery, 676
Bartlett, W. C.: Acute Cholecystitis: An Analysis of Sixty-One Cases, 119
Basal Cell Carcinoma of the Anus, J. G. Matt, and J. T. Dilger, 886
Basal Cell Carcinoma of the Anus: A Case Report, C. W. Fitchett, and J. P. Baker, 641
Beall, A. C., Jr.: See Overton, R. C.
Benoit, H. W., Jr., and Mayer, J. H. Jr.: Esophageal Perforations, 313
Neonatal Respiratory Problems Exclusive of Central Nervous System Causes, 1126
Berg, H. F., and Christopherson, W. M.: Selective Irradiation of Lymph Nodes by Interstitial Injection of Radioactive Colloidal Gold, 172
Bergner, R. P.: Anesthetic Complications in Pediatric Thoracic Surgical Patients, 187
Bickham, C. E., Jr.: Intravenous Cholecystography and Cholangiography using Cholografin, 1225
Biliary tract surgery, 615
Blair, E., Swan, H., and Virtue, R.: Clinical Hypothermia: A Study of the Ice-water Surface Immersion and Short-Wave Diathermy Rewarming Technics, 869
Blair, W. M.: Psoas Myositis or Fibrositis in the Differential Diagnosis of Lower Abdominal Disease, 505
Bleeding, gastrointestinal, 606
Block, bundle branch, 80
Boley, J. O.:
See Robinson, D. W.
Book Review, 265
Boli of ingested coconut, 1076
Bollack, C. G.: See Hermann, L. G.
Bone
fractures, 1005
oral epithelium, 194
Book Reviews
Atlas of General Surgery, J. R. Wilder, 362
Anatomy for Surgeons, Vol II, W. H. Hollingshead, 1234
Breast Cancer and Its Diagnosis and Treatment, E. F. Lewison, 264
Cancer Cells, E. V. Cowdry, 265
Diagnosis and Treatment of Vascular Disease (Angiology), S. S. Samuels, 1040
Fluid Balance Handbook for Practitioners, Wm. D. Snively, Jr., and M. J. Sweeney, 1234
Fractures of the Facial Skeleton, N. L. Rowe, 826
Joints of the Extremities: A Radiographic Study, R. W. Lewis, 265
Operative Technic in Specialty Surgery, W. H. Cole, 608
Plastic Repair of Genito-Urinary Defects, G. Bankoff, 1235
Scalpel-Men Who Made Surgery, A. Young, 1040
Stereoscopic Atlas of Human Anatomy, D. L. Bassett, 453
Transactions of American Goiter Association: 1955, 1040
Transplantation of Tissues: Cartilage, Bone, Fascia, Tendon, and Muscle, L. A. Peer, 264

Bossert, L. J., and Haydon, G. B.: Experiences with Vaginal Hysterectomy at the Cincinnati General Hospital: A Ten Year Study, 780
Bowel, adenomatous polyps, 964
Bowen, F. H.: Obstruction of the Small Bowel due to Boli of Ingested Coconut: A Report of Two Cases, 1076
Bowers, R. F.: Vena Cava Ligation: Advantages VS Disadvantages (Editorial), 359
Boyd, W. M.: Hypothermia in Hyperthyroidism: A Case Report, 857
Brabham, V. W.: See Wolfe, A. B.
Brackett, C. E., Jr.: See Williamson, W. P.

Branchiogenic malignancy, 975
Breast
  cancer, 942
  carcinoma, 897
Breck, L. W.: Intramedullary Fixation of Long Bone Fractures, 1005
Brief History of the Department of Surgery, University of Louisville, School of Medicine, D. P. Hall, 159
Bronchiectasis, abronchiectatic, 465
Bronchiectatic patient, 922
Brooker, R. M.: Spontaneous Perforation of the Esophagus with Report of a Case, 129
Brown, B. H.: See Brown, G. M., Jr.
Brown, G. M., Jr., and Brown, B. H.: Routine Operative Cholangiography: A Critical Five Year Review, 415
Brown, J. B.: Homo Skin Grafts from Postmortem Sources for Severe Burns (Editorial), 541
Brownlee, W. E.: See Higgins, G. A.
Bryant, J. R.: See Rubel, W. F.
Buente, L. C., McGrath, E. J., Pasquale, A., and Vetto, R. M.: One Stage Extraperiosteal Lucite Plombage in the Treatment of Pulmonary Tuberculosis, 725
Bundle branch block, 80
Burns
  caustic, of esophagus, 308
  homo skin grafts, 541
Butler, D. E., and Eichhorn, R.: Acute Pancreatitis with Necrosis and Perforation of Common Bile Duct: Case Report, 512
Buxton, R. W.: Application of Neck Gland Resections, 16

Calcified Subdural Hematomas in Childhood: Report of Two Cases, R. L. McLaurin, 736
Cancer
  thyroid, 847
  Ultraradical surgery, 451
Cannon, B. W.: Acute Spinal Injuries, 1108
Carcinoma
  anus, 641, 886
  papillary of thyroid, 399
  prostatic, 894
Carcinoma of the Breast: A Ten Year Appraisal, R. T. Myers, and J. H. Meredith, 897
Cardiospasm and Esophagus: An Experimental Study of the Esophagogastric Sphincter, S. R. Friesen, and D. R. Miller, 267
Careless Anesthesia as a Hazard to the Suture Line (Editorial), A. R. Koontz, 1038
Case for Hemigastrectomy and Vagotomy in Surgical Treatment of Duodenal Ulcer, E. D. Grady, 1052
Castleton, K. B.: Tissue Committees (Editorial), 929
Cervical spondylosis, 227
Charache, H.: Malignant Degeneration in Sebaceous Cysts, 482
Chest
  trauma, 382
  wall, repair of surgical defects, 139
Chloride and sodium determinations, 1117
Cholangiography
  routine operative, 415
  using cholografin, 1225
Cholecystitis
  acute, 119
  acute emphysematous, 664
  surgical therapy, 1137
Cholecystography, 1225
Cholecystostomy in Modern Surgery, C. Costello, 1079
Christopherson, W. M.: See Berg, H. F.
Clark, G. G.: See Silvis, R. S.
Clark, J. H.: Rational Treatment of Painful Vasomotor Disturbances Involving the Upper Extremities, 420
Clinical Experiences with Prefabricated Nylon Artery Grafts, W. S. Edwards, and J. S. Tapp, 1174
Clinical Hypothermia: A Study of the Ice-water Surface Immersion and Short-Wave Diathermy Rewarming Technics, E. Blair, H. Swan and R. Virtue, 869
Coarctation, aorta, 1168
Cohn, I.: Bacterial Factors in Strangulation Obstruction: Review of Experimental Work, 836
—— and Longacre, A. B.: Neomycin-Nystatin for Preoperative Preparation of the Colon, 301
Cole, W.: See Altemeier, W. A.
Colloidal gold, 172
Colon
  diverticulosis and diverticulitis, 683
  polyposis, 583
  polyps, 287

preoperative, 301
treatment of diverticulitis, 4
vesicocolic fistula, 645
Coloscopy, transabdominal, 153
Colostomy, 827
Combined Nerve and Tendon Injuries in the Hand and Forearm, V. E. Siler, 764
Comparison of Radical and Less Extensive Surgery in Cancer of the Breast, W. C. White, 942
Complete Rectal Prolapse: The Orr Treatment (Surgical Technic), J. B. Floyd, Jr., and R. Alley, 336
Competence of the Esophagogastric Sphincter in Hiatal Hernia; Some Experimental Observations, S. R. Friesen and D. R. Miller, 42
Controversial Branchiogenic Malignancy, W. C. Jones, C. C. Snyder, and L. W. Dowlen, 975
Cooley, D. A., DeBakey, M. E., and Creek, O., Jr.: Surgical Treatment of Aortic Aneurysms, 1043
Cooper, I. S.: Neurosurgical Alleviation of Parkinsonism, 1070
Cortex, adrenal, 56
Costello, C.: Cholecystostomy in Modern Surgery, 1079
Creech, O., Jr.: See Cooley, D. A.
Cryptorchidism, R. Lich, Jr., 198
Crystalloid solutions, 93
Curare, 156
Cyst
kidney, 573
omental dermoid, 652
sebaceous, 482

Daniel, D. S.: Hospital House Staff: Its History, Present Day Situation, and an Educational Program in a Private Hospital, 931
Davis, J. B.: Diverticulosis of the Jejunum: Report of a Case Simulating Gastric Carcinoma, 330
Davis, M. V.: Early and Late Management of Caustic Burns of the Esophagus, 308
Davis, W. B.: See Rubel, W. F.
DeBakey, M. E.: See Cooley, D. A.
Debridement, wounds, 543
Defects, chest wall, 139
Definitive Radical Resection of the Large Bowel for Recurrent Diverticulitis, G. H. Yeager, 611
Delayed Hemothorax due to Traumatic Rupture of the Internal Mammary Artery: A Case Report, P. T. McGannon, and H. A. Frank, 135
Demong, C. V.: See Grow, J. B.
Denervation, peripheral, 97
Department of Surgery, Residency Programs (Editorial), R. J. Noer, 262
Dermoid, omental cyst, 657
Dextran, 93
Diagnosis
adrenal cortical insufficiency, 1117
jaundice, 657
Diagnosis of Perforating Arteriosclerotic Abdominal Aortic Aneurysms: A Review of Cases, T. Farrar, and R. L. Sanders, 623
Diagnostic Peritoneal Tap, W. H. Moretz, and W. G. Erickson, 1095
Diagnostic urology, 236
Diamond, I.: See Wolfe, J. J.
Diaphragm, traumatic eventration, 1194
Diathermy, rewarming, 869
Dilatation, gastric, 1154
Dilger, J. T.: See Matt, J. G.
Dimond, E. G.: See Kittle, C. F.
Disease
abdominal, 505
peripheral arteriosclerotic, 1163
thrombo-occlusive, 428
Disk surgery, 752
Diverticula, pharyngo-esophageal, 880
Diverticulitis, 295, 611, 645
colon, 4, 683
Diverticulosis and Diverticulitis of the Colon, M. M. Zinninger, 683
Diverticulosis of the Jejunum, D. R. Jensen, 322
Diverticulosis of the Jejunum: Report of a Case Simulating Gastric Carcinoma, J. B. Davis, 330
Dixon, C. F.: Surgical Treatment of Diverticulitis of the Colon, 4
Dorst, S. E.: Foreword, University of Cincinnati College of Medicine, 673
Dowlen, L. E.: See Jones, W. C.
Duct, bile, 512
Duncan, J. T., Jr.: Rupture of the Small Intestine through the Intact Abdominal Wall Without Associated Intraperitoneal Injury, 1215
Duodenal ulcer, 1052
Durham, M. W., and Hart, W.: Epithelioma of the Jejunum, 602

Eade, G. G.: See Zech, R. K.
Early and Late Management of Caustic Burns of the Esophagus, M. V. Davis, 308
Eberhart, C.: Book Review, 1235
Editorials
Careless Anesthesia as a Hazard to the Suture Line, A. R. Koontz, 1038
Department of Surgery; Residency Programs, R. J. Noer, 262
Future of Antibiotic Treatment in Surgery, W. A. Altemeier, and Wm. Cole, 821
Has Curare been Maligned? P. H. Lorhan, 156
Homo Skin Grafts from Postmortem Sources for Severe Burns, J. B. Brown, 541
Law of Diminishing Returns as it Applies to Ultraradical Surgery for Cancer, H. K. Gray, 451
Massive Upper Gastrointestinal Bleeding, J. D. Martin, Jr., 606
Not For Ourselves Alone, C. W. Mayo, 1233
Progress in the Development of Arterial Prostheses, A. H. Blakemore, 1134
Tissue Committees, K. B. Castleton, 929
Vena Cava Ligation: Advantages VS Disadvantages, R. F. Bowers, 359

Edwards, W. S., and Tapp, J. S.: Clinical Experiences with Prefabricated Nylon Artery Grafts, 1174
Eichhorn, R.: See Butler, D. E.
Embryonic parathyroid tissue transplant, 1222
Emphysema, mediastinal, 567
Emphysematous cholecystitis, 664
Enteritis, 1149
Enzymatic Debridement of Wounds: A Preliminary Report, A. G. Packard Jr., 543
Epithelioma of the Jejunum, M. W. Durham, and W. Hart, 602
Epithelium, oral, 194
Erickson, W. G.: See Moretz, W. H.
Esophageal hiatal hernia, 668
Esophageal Perforations, H. W. Benoit, Jr., and J. H. Mayer, Jr., 313
Esophagitis, cardiospasm, 267
Esophagogastric sphincter, 42, 267
Esophagus
caustic burns, 308
spontaneous perforation, 129
Estes, W. L.: Five Year End Results in the Treatment of Inguinal Hernia, 948
Eventration, traumatic of diaphragm, 1194
Experience in the Surgical Management of Atrial Septal Defect and Pulmonic Stenosis Under Hypothermia, J. M. Salyer, C. S. Lyter, and B. E. Pollock, 1058
Experience with the Extended Postoperative Care of Congenital Megacolon, C. M. Lee, 705
Experiences with Frontal Lobotomy, E. G. Grantham, and L. H. Segerberg, 242
Experiences with Vaginal Hysterectomy at the Cincinnati General Hospital: A Ten Year Study, L. J. Bossert, and G. B. Haydon, 780
Experimental Study of Oral Epithelium Buried in Bone: Preliminary Report, J. J. Wolfe, and I. Diamond, 194
Experimental Vascular Grafts. X: Studies of Fresh Venous Autografts Treated with Hevea Latex or Plication, R. K. Zeach, L. M. Nyhus, G. G. Eade, and H. N. Harkins, 455
Extremity
amputation, 696
lower, longitudinal growth, 97

Familial Polyposis of the Colon, R. P. McBurney, and R. L. Sanders, 583
Farrar, T., and Sanders, R. L.: Diagnosis of Perforating Arteriosclerotic Abdominal Aortic Aneurysms: A Review of Cases, 623
Fascia lata patches, 217
Fatal Rebleeding Indices in Dogs Following Administration of Blood, Plasma, Dextran, and Crystalloid Solutions, C. L. Henry, G. C. Nelson, and W. F. Pipes, 93
Feminizing Tumors of the Adrenal Cortex, G. A. Higgins, W. E. Brownlee, and F. A. Mantz, Jr., 56
Femur fractures, 801
Fiberglas prosthesis, repair in chest wall, 139
Fibrillation, ventricular, 436
Fibromatosis, irradiation, 33
Fibrosarcoma, irradiation, 33
Fibrositis, 505

Fistula
gastrojejunocolic, 517
vesicocolic, 645
Fitchett, C. W., and Baker, J. P.: Basal Cell Carcinoma of the Anus: A Case Report, 641
Five Year End Results in the Treatment of Inguinal Hernia, W. L. Estes, Jr., 948
Floyd, J. B., Jr., and Alley, R.: Complete Rectal Prolapse: The Orr Suspension Treatment, 336
Forearm, nerve and tendon injuries, 764
Foreword, University of Cincinnati College of Medicine, S. E. Dorst, 673
Fractures
femur, 801
long bone, 1005
treatment, 485
Frank, H. A.: See McGannon, P. T.
Free Mattressed Fascia Lata Patches in the Repair of Large Abdominal Incisional Hernias, J. E. Hamilton, 217
Friesen, S. R., and Miller, D. R.:
Cardiospasm and Esophagitis: An Experimental Study of the Esophagogastric Sphincter, 267
Competence of the Esophagogastric Sphincter in Hiatal Hernia: Some Experimental Observations, 42
Future of Antibiotic Treatment in Surgery (Editorial), W. A. Altemeier, and Wm. Cole, 821

Galactose tolerance test, 657
Ganem, E. J.: Relationship of Symptoms to Prognosis in Advanced Prostatic Carcinoma, 894
Garlick, W. L.: Management of the Bronchiectatic Patient who Cannot be Considered a Candidate for Surgery, 922
Gastrectomy, total, 363
Gastric
dilatation, 1154
polyposis, 474
Gastrointestinal
bleeding, 606
hemorrhage, 1123
Gastrojejunocolic Fistula: One Stage Resection and Repair, J. M. Keshishian, and O. Gwathmey, 517
Gerwig, W. H., Jr.: Anorectal Complications of Pregnancy, 549
Giant Cyst of the Kidney: A Case Report, H. Jacobs, and H. C. Saltzstein, 573
Gillesby, W. J., and O'Reilly, C. M.: Acute Appendicitis in the Elderly Male, 1186
—— and Wheeler, J. R.: Acute Gastric Dilatation, 1154
Giuseffi, J., and McElhinney, T.: Suggestions on Technic in Through-and-Through Wire Closure of Abdominal Wounds, 746
Glands
adrenal, 904
nodular thyroid, 1014
resections, neck, 16
Gold, colloidal, 172
Grady, E. D.: Case for Hemigastrectomy and Vagotomy in Surgical Treatment of Duodenal Ulcer, 1052
Grafting, homologous, 428
Grafts
experimental, 455
homo skin, 541
nylon, 1174
Grantham, E. G., and Segerberg, L. H.: Experiences with Frontal Lobotomy, 242
Gray, H. K.: Law of Diminishing Returns as it Applies to Ultraradical Surgery for Cancer (Editorial), 451
Groth, R. C.: Ancient Anodynes: A History of Anesthesia Prior to 1750 (Historical), 345
Grow, J. B., Demong, C. V., and Rundles, W. R.: Necrotizing Panarteritis: A Complication of Repair of Coarctation of the Aorta, 1168
Growdon, J. H., and Stainton, R. M.: Acute Perforated Ulcer—An Analysis of Eighty-Seven Cases, 278
Gwathmey, O.: See Keshishian, J. M.

Hall, D. P.: Brief History of the Department of Surgery, University of Louisville School of Medicine, 159
Hamilton, J. E.: Free Mattressed Fascia Lata Patches in the Repair of Large Abdominal Incisional Hernias, 217
—— See Lewis, B.
Hand, tendon and nerve injuries, 764
Hanlon, C. R.: Surgical Treatment of Funnel Chest (Pectus Excavatum), 408
Hannon, K. M., and Smith, E. T.: Malignant Metastasis to the Lumbar Spine Simulating Disk Rupture: Report of Two Cases, 1019
Hardin, C. A.: Book Review, 264
—— Resection and Homologous Grafting

of the Terminal Aorta and Iliac Arteries for Thrombo-Occlusive Disease, 428
—— and Kittle, C. F.: Repair of Surgical Defects of the Chest Wall with Fiberglas Prosthesis (Surgical Technic), 139
Harkins, H. N.: See Stevenson, J. K., and Zech, R. K.
Hart, W.: See Durham, M. W.
Harter, J. S.: See Rubel, W. F.
Has Curare been Maligned? (Editorial), P. H. Lorhan, 156
Haslan, E. T.: See Litton, L. O.
Haydon, G. B.: See Bossert, L. J.
Head injuries, 528
Helmsworth, J. A.: Treatment of Wounds of the Heart: A Comparison of Methods and an Analysis of Results, 719
Hemangiopericytoma (Glomus Tumor) of the Thigh: Case Report, T. R. Stevenson, 393
Hematomas, subdural calcified, 736
Hemigastrectomy, 1052
Hemorrhage
following removal of gauze pack, 579
gastrointestinal, 1123
Hemothorax, 135
Hendrick, J. A.: See Knight, C. D.
Hendrick, J. W.: Treatment of Gastric Polyposis, 474
Hendricks, D.: See Robbins, J. J.
Henry, C. L., Nelson, G. C., and Pipes, W. F.: Fatal Rebleeding Indices in Dogs Following Administration of Blood, Plasma, Dextran, and Crystalloid Solutions, 93
Hernia
esophageal, 668
hiatal, 42
inguinal, 948
large abdominal incisional, 217
ventral, 789
Herrington, J. L.: Simultaneous Small and Large Bowel Obstruction Resulting from Chronic Regional Enteritis, 1149
Herrmann, L. G., and Bollack, C. G.: Treatment of Large Nerves at the time of Amputation of an Extremity: A Probable Cause for Persistent Pain, 696
Hiatal hernia, 42
Higgins, G. A.: Surgery of the Adrenal Glands, 904
—— Transabdominal Coloscopy (Surgical Technic) 153
—— Brownlee, W. E., and Mantz, F. A., Jr.: Feminizing Tumors of the Adrenal Cortex, 56
Homografting, arterial, 1163
Homologous grafting, 428
Homo Skin Grafts from Postmortem Sources for Severe Burns (Editorial), J. B. Brown, 541
Hooks, C. A.: See Thompson, I. M.
Horger, R. C.: See Wolfe, A. B.
Hospital House Staff: Its History, Present Day Situation, and an Educational Program in a Private Hospital, D. S. Daniel, 931
Hyperthyroidism, 857
Hypertrophic Pyloric Stenosis: A Clinical Analysis of Eighty-Seven Cases with Special Reference to Etiologic Factors, D. R. Miller, and S. R. Friesen, 108
Hypothermia
clinical, 869
general, 436
Hypothermia in Hyperthyroidism: A Case Report, W. M. Boyd, 857
Hysterectomy, vaginal, 780

Ileostomy, 827
Iliac arteries, 428
Indeterminant Pulmonary Cavity, P. W. Schafer, 925
Infundibulectomy, 80
Inguinal hernia, 948
Injuries
hand and forearm, 764
head, 528
intraperitoneal, 1215
spinal, 1108
vascular, 752
Intervertebral disk surgery, 752
Intestinal intubation, 598
Intra-abdominal conditions, urologic pathology, 372
Intracranial aneurysms, 100
Intramedullary Fixation of Long Bone Fractures, L. W. Breck, 1005
Intraperitoneal injury, 1215
Intravenous Cholecystography and Cholangiography using Cholografin, C. E. Bickham, Jr., 1225
Intubation, intestinal, 598
Irradiation Fibromatosis and Fibrosarcoma, D. W. Robinson, and J. O. Boley, 33
Irradiation, lymph nodes, 172

Jacobs, H., and Saltzstein, H. C.: Giant Cyst of the Kidney, 473

Jaffe, I. A.: Traumatic Rupture of the Liver with Severe Secondary Hemorrhage Following Removal of a Gauze Pack, 579
Jaundice, diagnosis, 657
Jeffords, J. V., and Stallworth, J. M.: Use of Refrigeration Anesthesia (Regional Hypothermia) for Amputation in Poor-Risk Patients with Peripheral Vascular Disease, 998
Jejunum
  diverticulosis, 322, 330
  epithelioma, 602
Jensen, D. R.: Diverticulosis of the Jejunum, 322
Johns, B.: See Knight, C. D.
Johnson, W. O.: Ovarian Tumors: A Five Year Study (1949–1954) in Four Louisville Hospitals, 179
Jones, W. C., Snyder, C. C., and Dowlen, L. W.: Controversial Branchiogenic Malignancy, 975
Jontz, J.: See Shumacker, H. B., Jr.

Kaden, V.: See Overton, R. C.
Kanthak, F. F.: Book Review, 826
Kartagener's Syndrome: Report of a Case Treated by Middle Lobe Lobectomy, T. C. Moore, and R. A. Silver, 595
Keshishian, J. M., and Gwathmey, O.: Gastrojejunocolic Fistula: One Stage Resection and Repair, 517
Kidney, cyst, 573
Kingsbury, H. A.: Surgical Treatment of Cancer of the Thyroid, 847
Kittle, C. F.: See Hardin, C. A.
—— Santos, E. M., and Dimond, E. G.: Persistent Right Bundle Branch Block due to Pulmonic Valvotomy and Infundibulectomy, 80
Knight, C. D., Johns, B., and Hendrick, J. A.: Surgical Therapy for Cholecystitis: Observations Based on a Review of Four Hundred and Seventeen Cases, 1137
Koontz, A. R.: Careless Anesthesia as a Hazard to the Suture Line (Editorial), 1038
Kraeft, N. H., and Linton, L. O.: Surgery in Pulmonary Tuberculosis, 1207
Krunkenberg Tumors, M. A. Rabe, 86
Kulowski, J.: Accidental Head Injuries in Occupants of Crash Motor Vehicles: A Report of Two Hundred and Seventy-Three Cases, 528

Late Sequellae of Traumatic Eventration of the Diaphragm and their Surgical Management, E. R. Maurer, 1194
Law of Diminishing Returns as it Applies to Ultraradical Surgery for Cancer (Editorial), H. K. Gray, 451
Lee, C. M., Jr.: Experience with the Extended Postoperative Care of Congenital Megacolon, 705
Lesions, obstructive of urinary tract, 630
Letton, A. H.: Book Review, 1040, 1041, 1234
Lewis, B., and Hamilton, J. E.: Acute Perforated Peptic Ulcers, 204
Lewis, J. E., Jr.: See Starkloff, G. B.
Lich, R., Jr.: Cryptorchidism, 198
Ligation, vena cava, 359
Linton, L. O.: See Kraeft, N. H.
Litton, L. O., and Haslam, E. T.: Longitudinal Growth of Lower Extremity Following Experimental Partial Peripheral Denervation, 97
Liver, rupture, 579
Lobectomy, middle lobe, 595
Lobotomy, frontal, 242
Longacre, A. B.: See Cohn, I., Jr.
Longitudinal Growth of Lower Extremity Following Experimental Partial Peripheral Denvervation, L. O. Litton, and E. T. Haslam, 97
Lorhan, P. H.: Has Curare been Maligned? (Editorial), 156
Lucite plombage, pulmonary tuberculosis, 725
Lyter, C. S.: See Salyer, J. M.:

Macgregor, C. A., and Munro, G. A.: Accuracy in the Diagnosis of Jaundice with Particular Reference to the Intravenous Galactose Tolerance Test, 657
Mack, J. R.: Major Vascular Injuries Incident to Intervertebral Disk Surgery, 752
MacQuigg, R. E.: Abronchiectatic Bronchiectasis: Recognition of Surgical Pneumonitis, 465
Mahorner, H.: Advances in Technical Aspects of Biliary Tract Surgery, 615
Major, R. H.: Tribute to Doctor T. G. Orr, Sr., 1
Major Vascular Injuries Incident to Intervertebral Disk Surgery, J. R. Mack, 752
Malignancy, branchiogenic, 975

Malignant Degeneration in Sebaceous Cysts, H. Charache, 482
Malignant Metastasis to the Lumbar Spine Simulating Disk Rupture: Report of Two Cases, K. M. Hannon, and E. T. Smith, 1019
Mammary artery, delayed hemothorax, 135
Management
  colostomy, 827
  ileostomy, 827
  surgical
    gastrointestinal hemorrhage, 1123
    traumatic eventration of diaphragm, 1194
Management of Intracranial Aneurysms of the Anterior Communicating Artery, W. P. Williamson, and C. E. Brackett, Jr., 100
Management of Spontaneous Pneumothorax, W. F. Rubel, J. S. Harter, J. R. Bryant, and W. B. Davis, 211
Management of the Bronchiectatic Patient who Cannot be Considered a Candidate for Surgery, W. L. Garlick, 922
Mantz, F. A., Jr.: See Higgins, G. A.
Martin, J. D., Jr.:
  Book Review, 362
  Massive Upper Gastrointestinal Bleeding (Editorial), 606
  Vesicocolic Fistula Complicating Diverticulitis of the Colon, 645
Mastectomy, 150
Matt, J. G., and Dilger, J. T.: Basal Cell Carcinoma of the Anus, 886
Maurer, E. R.: Late Sequellae of Traumatic Eventration of the Diaphragm and their Surgical Management, 1194
Mayer, J. H., Jr.: See Benoit, H. W., Jr.
Mayo, C. W.: Not for Ourselves Alone (Editorial), 1233
McBurney, R. P.:
  Pharyngo-Esophageal Diverticula, 880
  Repair of Esophageal Hiatal Hernia Utilizing Allison's Principle Through an Abdominal Approach (Surgical Technic), 668
—— and Sanders, R. L.: Familial Polyposis of the Colon, 583
McElhinney, T.: See Giuseffi, J.
McGannon, P. T., and Frank, H. A.: Delayed Hemothorax Due to Rupture of the Internal Mammary Artery: A Case Report, 135
McGrath, E. J.: See Buente, L. C.
McLaurin, R. L.: Calcified Subdural Hematomas in Childhood: Report of Two Cases, 736
Mead, W. R.: See Rigdon, H. L.
Mediastinal Emphysema and Bilateral Pneumothorax Following Surgery of the Neck, L. C. Rollins, and D. H. Poer, 567
Megacolon, congenital, 705
Meningocele, anterior sacral, 554
Meredith, J. H.: See Myers, R. T.
Metastasis, malignant to lumbar spine, 1019
Miller, D. R., and Friesen, S. R.: Hypertrophic Pyloric Stenosis: A Clinical Analysis of Eighty-Seven Cases with Special Reference to Etiologic Factors, 108
Moore, T. C., and Silver, R. A.: Kartagener's Syndrome: Report of a Case Treated by Middle Lobe Lobectomy, 595
Moretz, W. H., and Erickson, W. G.: Diagnostic Peritoneal Tap, 1095
Munro, G. A.: See Macgregor, C. A.
Myers, R. T., and Meredith, J. H.: Carcinoma of the Breast, A Ten Year Appraisal, 897
Myositis, psoas, 505

Navratil, J.: Prevention and Treatment of Ventricular Fibrillation in General Hypothermia, 436
Neck
  gland resections, 16
  surgery, 567
Necrosis of common bile duct, 512
Necrotizing Panarteritis: A Complication of Repair of Coarctation of the Aorta, J. B. Grow, C. V. Demong, and W. R. Rundles, 1168
Nelson, G. C.: See Henry, C. L.
Neomycin-Nystatin for Preoperative Preparation of the Colon, I. Cohn, Jr., and A. B. Longacre, 301
Neonatal Respiratory Problems Exclusive of Central Nervous System Causes, H. W. Benoit, Jr., and J. H. Mayer, Jr., 1126
Neurosurgical Alleviation of Parkinsonism, I. S. Cooper, 1070
Nicolson, W. P.: Book Review, 608
Nodes, lymph, 172
Nodular thyroid gland, surgery, 1014
Noer, R. J.: Department of Surgery: Residency Programs (Editorial), 262

Not for Ourselves Alone, (Editorial), C. W. Mayo, 1233
Nyhus, L. M.: See Zech, R. K.
Nylon artery grafts, 1174

Obstruction
  bowel, 1149
  respiratory, 258
  strangulation, 836
Obstruction of the Small Bowel Due to Boli of Ingested Coconut: A Report of Two Cases, F. H. Bowen, 1076
Obstructive lesions, urinary tract, 630
Omental Dermoid Cyst, J. O. Warfield, Jr., 652
One Stage Extraperiosteal Lucite Plombage in the Treatment of Pulmonary Tuberculosis, L. C. Buente, E. J. McGrath, A. Pasquale, and R. M. Vetto, 725
Operative cholangiography, 415
O'Reilly, C. M.: See Gillesby, W. J.
Orr suspension treatment, rectal prolapse, 336
Orr, T. G., Jr.:
  Book Review, 265, 453
  Technic of Wound Closure Following Radical Mastectomy (Surgical Technic), 150
Ovarian Tumors: A Five Year Study (1949–1954) in Four Louisville Hospitals, W. O. Johnson, 179
Overton, R. C., Self, M. M., Kaden, V., and Beall, A. C., Jr.: An Unusual Complication of Intestinal Intubation: Case Report, 598

Pack, G. T.: Sarcomas of the Soft Somatic Tissues, 983
Packard, A. G., Jr.: Enzymatic Debridement of Wounds, A Preliminary Report, 543
Pain, amputation of extremity, 696
Panarteritis, necrotizing, 1168
Pancreatitis, acute, 512
Papillary Carcinoma of the Thyroid, R. Woodruff, 399
Parathyroid tissue transplant, 1222
Parkinsonism, neurosurgical alleviation, 1070
Pasquale, A.: See Buente, L. C.
Patients, pediatric thoracic surgical, 187
Pectus excavatum, surgical treatment, 408
Pediatric thoracic surgical patients, 187
Peptic ulcers, 204, 278
Perforation
  common bile duct, 512
  esophageal, 313
  spontaneous, esophagus, 129
Peripheral
  arteriosclerotic disease, 1163
  denervation, 97
  vascular diseases, amputation, 998
Peritoneal tap, diagnostic, 1095
Persistent Right Bundle Branch Block Due to Pulmonic Valvotomy and Infundibulectomy, C. F. Kittle, E. M. Santos, and E. G. Dimond, 80
Pharyngo-Esophageal Diverticula, R. P. McBurney, 880
Pinck, R. L.: See Thompson, J. E.
Pipes, W. F.: See Henry, C. L.
Pirkey, E. L., and Undem, D.: Roentgen Interpretation of Abnormal Gas Shadows seen on Plain Roentgenograms of the Abdomen, 249
Plantar Surfaces and the Weight Bearing Problem, D. W. Robinson, 442
Plasma, 93
Pneumonitis, surgical, 465
Pneumoperitoneum, spontaneous, 525
Pneumothorax
  following surgery of neck, 567
  spontaneous, 211
Poer, D. H.: See Rollins, L. C.
Pollock, B. E.: See Salyer, J. M.
Polyposis
  colon, 583
  gastric, 474
Polyps
  adenomatous of large bowel, 964
  colon and rectum, 287
Pregnancy
  abdominal, 1029
  anorectal complications, 549
Prevention and Management of Complications of Ileostomy and Colostomy, A. H. Storck, 827
Prevention and Treatment of Ventricular Fibrillation in General Hypothermia, J. Navratil, 436
Progress in the Development of Arterial Prostheses (Editorial), A. H. Blakemore, 1134
Prolapse, rectal, 336
Prostatic carcinoma, 894
Prostheses

arterial, 1134
fiberglas in chest wall repair, 139
Psoas Myositis or Fibrositis in the Differential Diagnosis of Lower Abdominal Disease, W. M. Blair, 505
Pulmonary
cavity, 925
tuberculosis, 1207
lucite plombage, 725
Pulmonic
stenosis, 1058
valvotomy, 80
Pyloric stenosis, 108

Rabe, M. A.: Krukenberg Tumors, 86
Radiation Hazard in Diagnostic Urology, J. J. Robbins, and D. Hendricks, 236
Radiation Hazards in the Practice of Surgery, C. M. Barrett, and E. L. Sanger, 676
Radical
mastectomy, 150
surgery, cancer of breast, 942
Radioactive colloidal gold, 172
Ransdell, H. T., Jr.: Tracheostomy in Absence of High Respiratory Obstruction, 258
Rational Treatment of Painful Vasomotor Disturbances Involving the Upper Extremities, J. H. Clark, 420
Read, G. T.: Treatment of Chest Trauma, 382.
Rectal prolapse, 336
Rectum, polyps, 287
Refrigeration anesthesia, 998
Reichert, F. L.: The Treatment of Preoperative and Postoperative Stress, 317
Relationship of Symptoms to Prognosis in Advanced Prostatic Carcinoma, E. J. Ganem, 894
Repair, coarctation of aorta, 1168
Repair of Esophageal Hiatal Hernia Utilizing Allison's Principle Through an Abdominal Approach (Surgical Technic), R. P. McBurney, 668
Repair of Surgical Defects of the Chest Wall with Fiberglas Prosthesis (Surgical Technic), C. A. Hardin, and C. F. Kittle, 139
Resection and Homologous Grafting of the Terminal Aorta and Iliac Arteries for Thrombo-Occlusive Disease, C. A. Hardin, 428
Resections, neck gland, 16
Residency programs, department of surgery, 262
Respiratory
obstruction, 258
problems, neonatal, 1126
Richards, R. C., and Woolf, C.: Solitary Polyps of the Colon and Rectum: A Study of Inherited Tendency, 287
Riddle, L. R.: See Silvis, R. S.
Rigdon, H. L., and Mead, W. R.: Successful Embryonic Parathyroid Tissue Transplant for the Treatment of Intractable Postoperative Parathyroid Tetany, 1222
Robbins, J. J., and Hendricks, D.: Radiation Hazard in Diagnostic Urology, 236
Robinson, D. W.: Plantar Surfaces and the Weight Bearing Problem, 442
—— and Boley, J. O.: Irradiation Fibromatosis and Fibrosarcoma, 33
Roentgen Interpretation of Abnormal Gas Shadows seen on Plain Roentgenograms of the Abdomen, E. L. Pirkey, and D. Undem, 249
Rollins, L. C., and Poer, D. H.: Mediastinal Emphysema and Bilateral Pneumothorax Following Surgery of the Neck, 567
Routine Operative Cholangiography: A Critical Five Year Review, G. M. Brown, Jr., and B. H. Brown, 415
Rubel, W. F., Harter, J. S., Bryant, J. R., and Davis, W. B.: Management of Spontaneous Pneumothorax, 211
Rundles, W. R.: See Grow, J. B.
Rupture of Abdominal Aortic Aneurysm into Inferior Vena Cava: Successful Surgical Repair, H. B. Shumacker, Jr., and J. Jontz, 1182
Rupture
liver, 579
traumatic, internal mammary gland, 135
Rupture of the Small Intestine Through the Intact Abdominal Wall Without Associated Intraperitoneal Injury, J. T. Duncan, Jr., 1215
Russin, L. A.: See White, E. H.

Sacral meningocele, 554
Saltzstein, H. C.: See Jacobs, H.
Salyer, J. M., Lyter, C. S., and Pollock, B. E.: Experience in the Surgical Management of Atrial Septal Defect and

Pulmonic Stenosis under Hypothermia, 1058
Sanders, R. L.: See McBurney, R. P., Farrar, T.
Sanger, E. L.: See Barrett, C. M.
Santos, E. M.: See Kittle, C. F.
Sarcomas of the Soft Somatic Tissues, G. T. Pack, 983
Schafer, P. W.: Indeterminant Pulmonary Cavity, 925
Schneewind, J. H.: Spontaneous Pneumoperitoneum, 525
Sebaceous cysts, malignant degeneration, 482
Segerberg, L. H.: See Grantham, E. G.
—— Spinal Cord Involvement due to Cervical Spondylosis, 227
Selective Irradiation of Lymph Nodes by Interstitial Injection of Radioactive Colloidal Cold, H. F. Berg, and W. M. Christopherson, 172
Self, M. M.: See Overton, R. C.
Septal defect, atrial, 1058
Shumacker, H. B., Jr., and Jontz, J.: Rupture of Abdominal Aortic Aneurysm into Inferior Vena Cava: Successful Surgical Repair, 1182
Sigmoid diverticulitis, 295
Siler, V. E.: Combined Nerve and Tendon Injuries in the Hand and Forearm, 764
Silver, R. A.: See Moore, T. C.
Silvis, R. S., Riddle, L. R., and Clark, G. G.: Anterior Sacral Meningocele, 554
Simultaneous Small and Large Bowel Obstruction Resulting from Chronic Regional Enteritis, J. L. Herrington, 1149
Smith, E. T.: See Hannon, K. M.
Snyder, C. C.: See Jones, W. C.
Sodium and chloride determinations, 1117
Solitary Polyps of the Colon and Rectum: Study of Inherited Tendency, R. C. Richards, and C. Woolf, 287
Some Unusual Obstructive Lesions of the Urinary Tract, W. S. Strode, 630
Spencer, J. R., and Wierman, W. H.: Urinary Sodium and Chloride Determinations as an Aid in the Diagnosis of Adrenal Cortical Insufficiency, 1117
Sphincter, esophagogastric, 42, 267
Spinal Cord Involvement due to Cervical Spondylosis, L. H. Segerberg, 227
Spine, lumbar, malignant metastasis, 1019
Spinal injuries, 1108
Spondylosis, cervical, 227
Spontaneous Perforation of the Esophagus with Report of a Case, R. M. Brooker, 129
Spontaneous Pneumoperitoneum, J. H. Schneewind, 525
Spontaneous pneumothorax, 211
Stainton, R. M.: See Growdon, J. H.
Stallworth, J. M.: See Jeffords, J. V.
Starkloff, G. B., and Lewis, J. E., Jr.: Surgical Treatment of Sigmoid Diverticulitis, 295
Stenosis
  pulmonic, 1058
  pyloric, 108
Stevenson, J. K., and Harkins, H. N.: Total Gastrectomy: Analysis of Twenty-Five Operations Performed at a County Hospital, 363
Stevenson, J. M.: Ventral Hernias, 789
Stevenson, T. R.: Hemangiopericytoma (Glomus Tumor) of the Thigh: Case Report, 393
Stewart, M. J.: Twenty-Five Years of Progress in the Treatment of Fractures, 485
Storck, A. H.: Prevention and Management of Complications of Ileostomy and Colostomy, 827
Stress, preoperative and postoperative, 317
Strode, W. S.: Some Unusual Obstructive Lesions of the Urinary Tract, 630
Successful Embryonic Parathyroid Tissue Transplant for the Treatment of Intractable Postoperative Parathyroid Tetany, H. L. Rigdon, and W. R. Mead, 1222
Suggestions on Technic in Through-and-Through Wire Closure of Abdominal Wounds, J. Giuseffi, and T. McElhinney, 746
Supracondylar Fractures of the Femur Treated by Internal Fixation with Immediate Knee Motion, E. H. White, and L. A. Russin, 801
Surgery
  antibiotic treatment, 821
  biliary tract, 615
  bronchiectatic patient, 922
  disk, 752
  modern, cholecystostomy, 1079

neck, 567
radiation hazards, 676
radical, cancer of breast, 942
ultraradical for cancer, 451
Surgery in Pulmonary Tuberculosis, N. H. Kraeft, and L. O. Linton, 1207
Surgery of the Adrenal Glands, G. A. Higgins, 904
Surgery of the Nodular Thyroid Gland, G. E. Twente, 1014
Surgical defect, chest wall, 139
Surgical management
atrial septal defect, 1058
traumatic eventration of diaphragm, 1194
Surgical Management of Massive Gastrointestinal Hemorrhage of Undetermined Origin, C. S. White, and C. S. White, Jr., 1123
Surgical pneumonitis, 465
Surgical Technic
Repair of Esophageal Hiatal Hernia Utilizing Allison's Principle Through an Abdominal Approach, R. P. McBurney, 668
Repair of Surgical Defects of the Chest Wall with Fiberglas Prosthesis, C. A. Hardin, and C. F. Kittle, 139
Technic of Wound Closure Following Radical Mastectomy, T. G. Orr, Jr., 150
Transabdominal Coloscopy, G. A. Higgins, 153
Surgical Therapy for Cholecystitis: Observations Based on a Review of Four Hundred and Seventeen Cases, C. D. Knight, B. Johns, and J. A. Hendrick, 1137
Surgical Treatment of Aortic Aneurysms, D. A. Cooley, M. E. DeBakey, and O. Creech, Jr., 1043
Surgical Treatment of Cancer of the Thyroid, H. A. Kingsbury, 847
Surgical Treatment of Diverticulitis of the Colon, C. F. Dixon, 4
Surgical treatment, duodenal ulcer, 1052
Surgical Treatment of Funnel Chest (Pectus Excavatum), C. R. Hanlon, 408
Surgical Treatment of Sigmoid Diverticulitis, G. B. Starkloff, and J. E. Lewis, Jr., 295
Swan, H.: See Blair, E.
Syndrome, Kartagener's, 595

Tapp, J. S.: See Edwards, W. S.
Technic of Wound Closure Following Radical Mastectomy (Surgical Technic) T. G. Orr, Jr., 150
Technics, diathermy rewarming, 869
Tendon injuries, hand and forearm, 764
Tetany, postoperative parathyroid, 1222
Therapy, surgical, for cholecystitis, 1137
Thigh, hemangiopericytoma, 393
Thompson, I. M., and Hooks, C. A.: Urologic Significance of Imperforate Anus, 1112
Thompson, J. E.: See Austin, D. J.
—— and Pinck, R. L.: Adenomatous Polyps of the Large Bowel: Their Significance to the Surgeon, 964
Thrombo-occlusive disease, 428
Thyroid
cancer, 847
gland, surgery, 1014
papillary carcinoma, 399
Tice, G. M.: Book Review, 265
Tissue Committees (Editorial), K. B. Castelton, 929
Tissue transplant, 1222
Total Gastrectomy: Analysis of Twenty-Five Operations Performed at a County Hospital, J. K. Stevenson, and H. N. Harkins, 363
Tracheostomy in Absence of High Respiratory Obstruction, H. T. Ransdell, Jr., 258
Transplant, tissue, 1222
Trauma, chest, 382
Traumatic Rupture of the Liver with Severe Secondary Hemorrhage Following Removal of a Gauze Pack, I. A. Jaffe, 579
Transabdominal Coloscopy (Surgical Technic), G. A. Higgins, 153
Treatment
antibiotic in surgery, 821
fractures, 485
Orr suspension for rectal prolapse, 336
rational vasomotor disturbances, 420
surgical
aortic aneurysms, 1043
cancer of thyroid, 847
diverticulitis of colon, 4
pectus excavatum, 408
sigmoid diverticulitis, 295
Treatment of Gastric Polyposis, J. W. Hendrick, 474
Treatment of Chest Trauma, G. T. Read, 382

Treatment of Large Nerves at the time of Amputation of an Extremity: A Probable Cause for Persistent Pain, L. G. Herrmann, and C. G. Bollack, 696
Treatment of Peripheral Arteriosclerotic Disease by Arterial Homografting, D. J. Austin, and J. E. Thompson, 1163
Treatment of Preoperative and Postoperative Stress, F. L. Reichert, 317
Treatment of Wounds of the Heart: A Comparison of Methods and an Analysis of Results, J. A. Helmsworth, 719
Tribute to Dr. T. G. Orr, Sr., R. H. Major, 1
Tumors
  adrenal cortex, 56
  Krukenberg, 86
  ovarian, 179
Tuberculosis, pulmonary, 725, 1207
Twente, G. E.: Surgery of the Nodular Thyroid Gland, 1014
Twenty-Five Years of Progress in the Treatment of Fractures, M. J. Stewart, 485

Ulcer
  duodenal, 1052
  peptic, 204, 278
Undem, D.: See Pirkey, E. L.
University of Cincinnati College of Medicine, 673
University of Louisville School of Medicine, 159
Unusual Complication of Intestinal Intubation: Case Report, R. C. Overton, M. M. Self, V. Kaden, and A. C. Beall, Jr., 598
Urinary Sodium and Chloride Determinations as an Aid in the Diagnosis of Adrenal Cortical Insufficiency, J. R. Spencer, and W. H. Wierman, 1117
Urinary tract, obstructive lesions, 630
Urologic Pathology Mimicking Intra-Abdominal Conditions, C. L. Wilson, and M. C. Wilson, 372
Urologic Significance of Imperforate Anus, I. M. Thompson, and C. A. Hooks, 1112
Urology, diagnostic, 236
Use of Refrigeration Anesthesia (Regional Hypothermia) for Amputation in Poor-Risk Patients with Peripheral Vascular Diseases, J. V. Jeffords, and J. M. Stallworth, 998

Vaginal hysterectomy, 780
Vagotomy, 1052
Valvotomy, pulmonic, 80
Vascular
  diseases, 998
  grafts, experimental, 455
  injuries, 752
Vasomotor disturbances, 420
Vena cava, 1182
Vena Cava Ligation: Advantages VS Disadvantages (Editorial), R. F. Bowers, 359
Venous autografts, 455
Ventral Hernias, J. M. Stevenson, 789
Ventricular fibrillation in general hypothermia, 436
Vesicocolic Fistula Complicating Diverticulitis of the Colon, J. D. Martin, Jr., 645
Vetto, R. M.: See Buente, L. C.
Virtue, R.: See Blair, E.

Warfield, J. O., Jr.: Omental Dermoid Cyst, 652
Wheeler, J. R.: See Gillesby, W. J.
White, C. S., and White, C. S., Jr.: Surgical Management of Massive Gastrointestinal Hemorrhage of Undetermined Origin, 1123
White, E. H., and Russin, L. A.: Supracondylar Fractures of the Femur Treated by Internal Fixation with Immediate Knee Motion, 801
White, W. C.: Comparison of Radical and Less Extensive Surgery in Cancer of the Breast, 942
Wierman, W. H.: See Spencer, J. R.
Williamson, W. P., and Brackett, C. E., Jr.: Management of Intracranial Aneurysms of the Anterior Communicating Artery, 100
Wilson, C. L., and Wilson, M. C.: Urologic Pathology Mimicking Intra-Abdominal Conditions, 373
Wilson, J. P.: Book Review, 1040
Wilson, M. C.: See Wilson, C. L.
Wolfe, A. B., Brabham, V. W., and Horger, R. C.: Abdominal Pregnancy: Report of Four Cases, 1029
Wolfe, J. J., and Diamond, I.: Experimental Study of Oral Epithelium Buried in Bone: Preliminary Report, 194

Woodruff, R.: Papillary Carcinoma of the Thyroid, 399
Woolf, C.: See Richards, R. C.
Wounds
abdominal, 746
closure, 150
debridement, 543
heart, 719

Yeager, G. H.: Definitive Radical Resection of the Large Bowel for Recurrent Diverticulitis, 611
Yedinak, P. R.: Acute Emphysematous Cholecystitis, 664

Zech, R. K., Nyhus, L. M., Eade, G. G., and Harkins, H. N.: Experimental Vascular Grafts. X: Studies of Fresh Venous Autografts Treated with Hevea Latex or Plication, 455
Zinninger, M. M.: Diverticulosis and Diverticulitis of the Colon, 683

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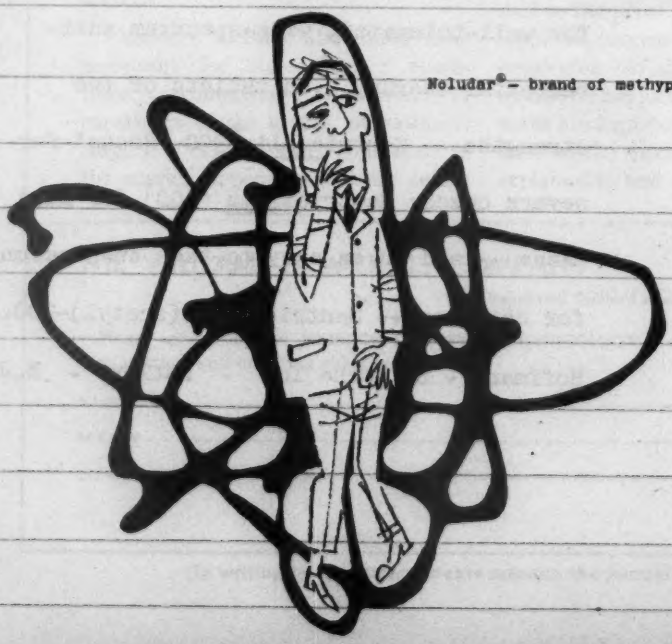

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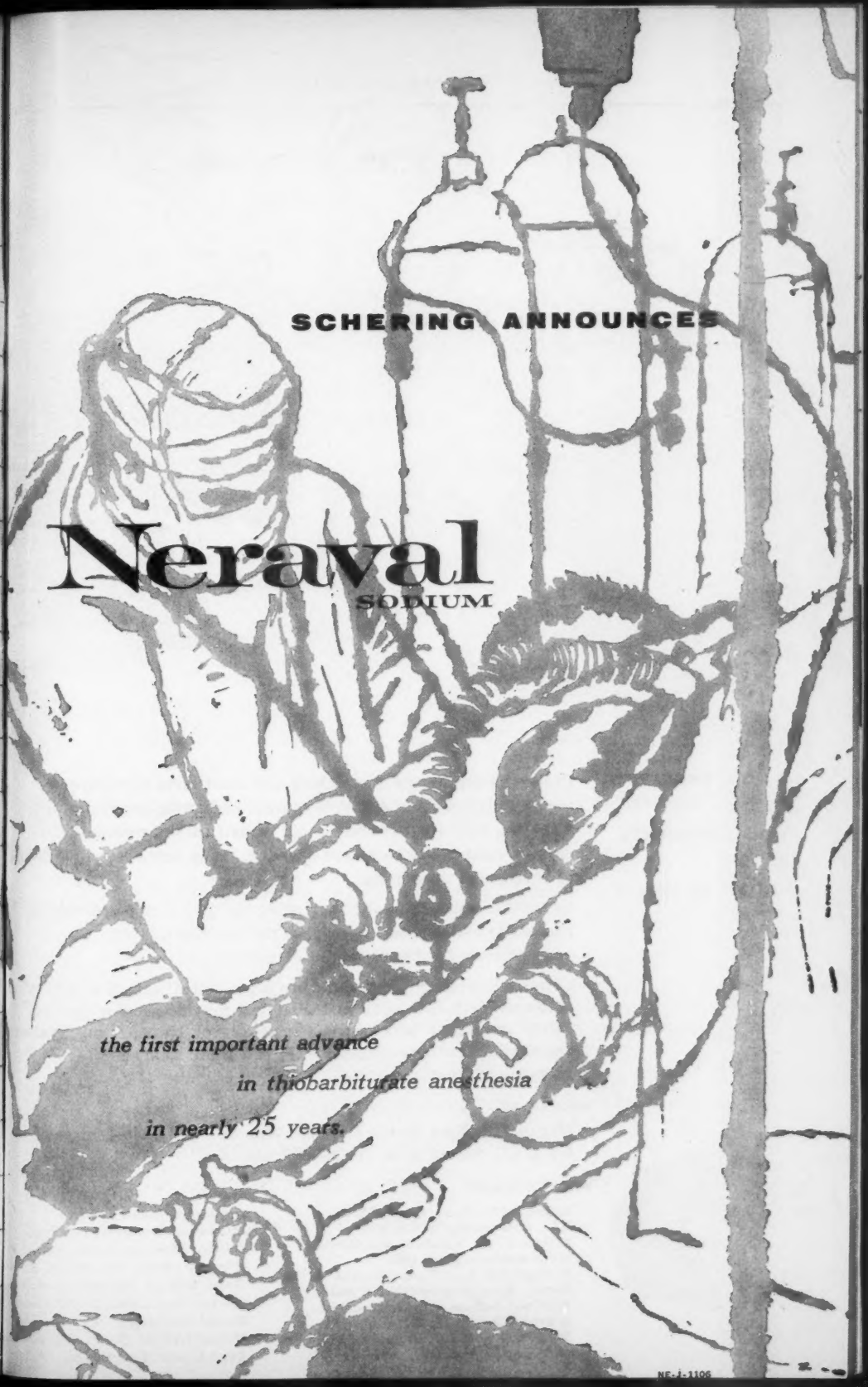
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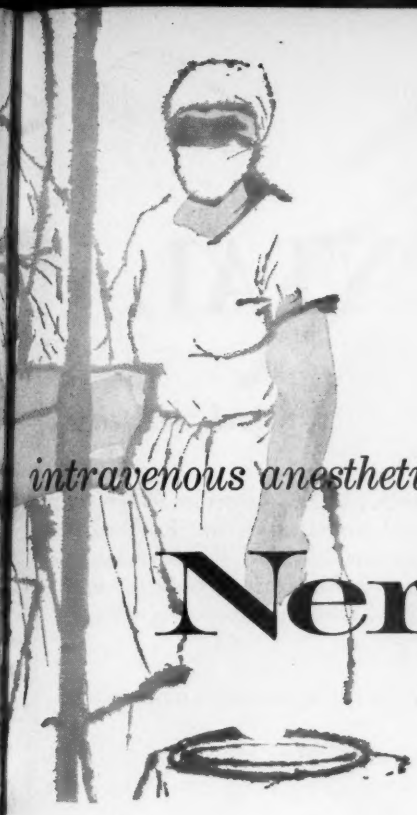

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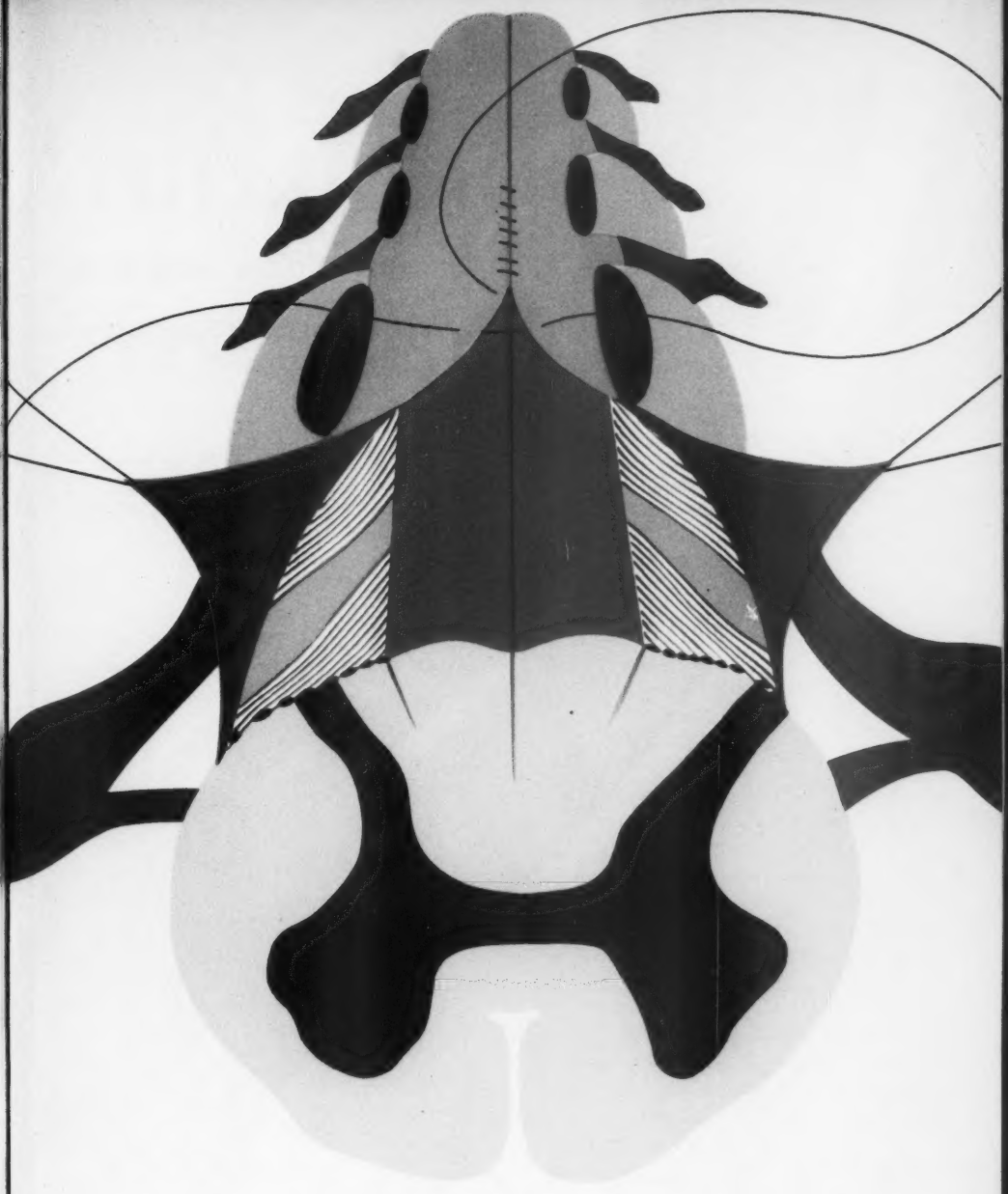

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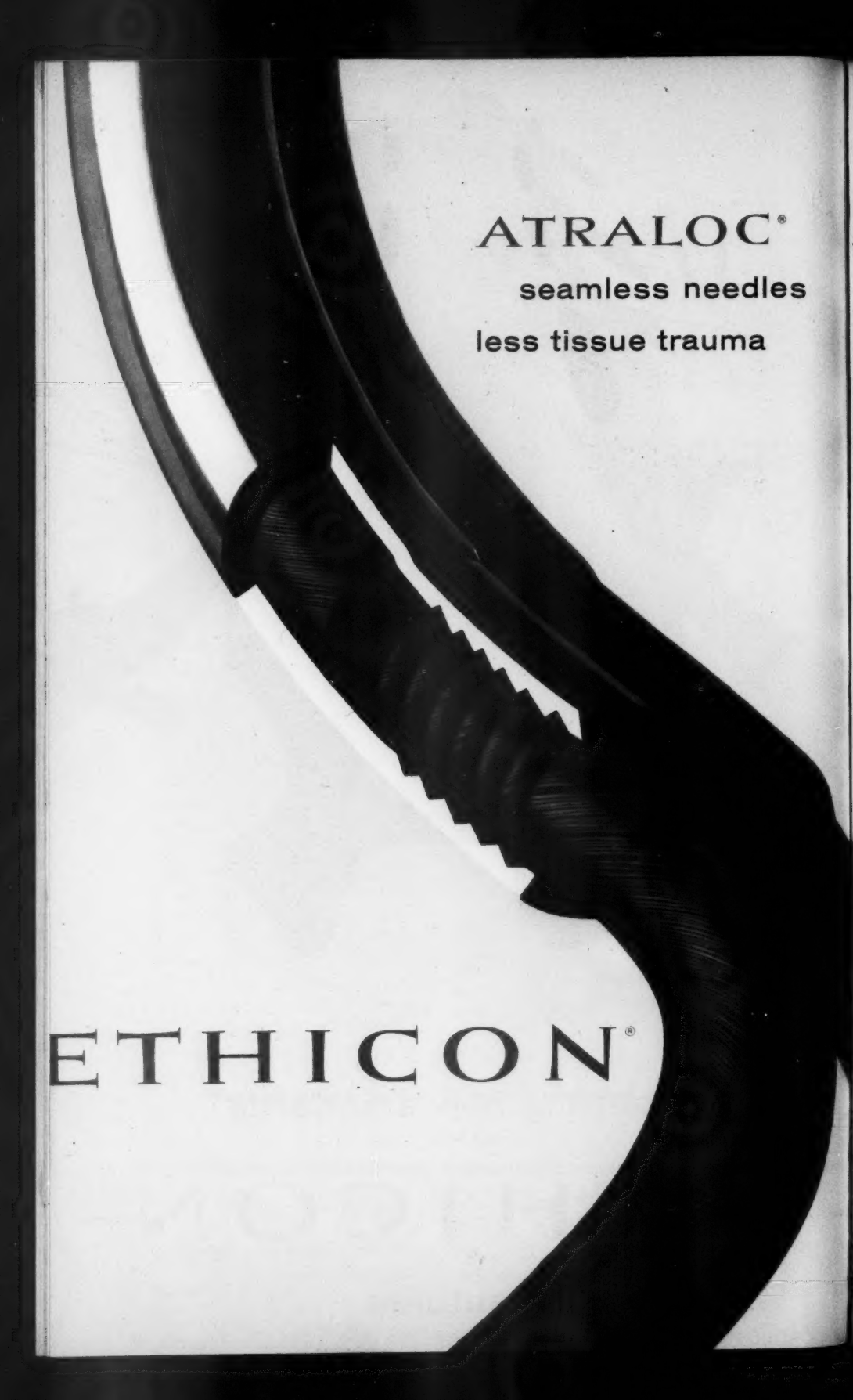
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